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EPIDEMIC (LETHARGIC) ENCEPHALITIS

CULTURAL AND EXPERIMENTAL STUDIES. PRELIMINARY COMMUNICATION *

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The following report presents evidence believed to be confirmatory of the results of Loewe and Strauss [1] with respect to the filtrability of the virus obtained from cases of epidemic encephalitis and the specific nature of the minute filtrable organism cultivated with special methods by these investigators.

Loewe and Strauss inoculated rabbits and monkeys with material from cases of epidemic (lethargic) encephalitis, following which a large percentage of the animals died, the central nervous system showing typical lesions of encephalitis. The materials injected were blood, spinal fluid, and Berkefeld [2] filtrates of nasal washings, also Berkefeld filtrates of the central nervous system and nasopharyngeal mucous membranes removed at necropsy. Their results demonstrated an infectious agent in the blood and spinal fluid and in Berkefeld filtrates of these materials from cases of encephalitis. It was found that the infectious agent is a filtrable one. Berkefeld filtrates of the brains of these animals caused the same disease when injected into other rabbits and monkeys. This virus was passed through many series of animals.

They were also able to cultivate on ascitic fluid tissue medium (as perfected by Noguchi [3]) a minute organism similar in morphologic,

* From the Laboratories of Columbia Hospital. This investigation was carried on with funds generously supplied by the Milwaukee Association of Commerce from a fund collected by them for the purpose of medical investigation. Thanks are gratefully extended to Dr. H. V. Ogden for his stimulating influence during the investigation.

1. Lowe, L., Hirshfeld, S., and Strauss, I.: Studies in Epidemic Encephalitis (Encephalitis Lethargica), Jour. Infect. Dis. **25**:378 (Nov.) 1919; New York M. J. **109**:772, 1919. Loewe, L., and Strauss, I.: Etiology of Epidemic (Lethargic) Encephalitis, J. A. M. A. **73**:1056 (Oct. 4) 1919; The Diagnosis of Epidemic Encephalitis, J. A. M. A. **74**:1373 (May 15) 1920; Proceedings of the New York Path. Soc. **20**:18, 1920; Studies in Epidemic (Lethargic) Encephalitis, Cultural Studies, J. Infect. Dis. **27**:250 (Sept.) 1920.

2. Mandler clay filters were also used by these investigators.

3. Noguchi, Hideyo: J. Exper. Med. **14**:99, 1911.

staining and cultural characteristics to the so-called "globoid bodies" recovered by Flexner and Noguchi [4] from cases of epidemic poliomyelitis. The organism of Loewe and Strauss is also a filter passer, and cultures of it injected intracranially into monkeys and rabbits produced the typical lesions of epidemic encephalitis in the central nervous system, resulting in death in most of the inoculated animals. The same micro-organism, with the same selective requirements for growth, was recovered from the brains of these animals.

This micro-organism differs from that of Flexner and Noguchi in "virulence, occurrence, and particularly in the ability to infect rabbits." It was also cultivated from the spinal fluid of epidemic encephalitis. Cultures infected rabbits and caused typical microscopic lesions, whereas monkeys were "relatively refractory." Flexner and Noguchi did not succeed in cultivating their organism from the spinal fluid of cases of poliomyelitis, and rabbits could not be infected with it, whereas monkeys were readily infected.

Levaditi and Harvier [5] also later demonstrated the relationship of a filtrable virus to epidemic encephalitis. With material from cases of this disease, passed through Berkefeld filters, they caused typical lesions in rabbits, monkeys and guinea-pigs, and they passed this virus through many series of animals, but they have not reported the cultivation of a filtrable organism.

McIntosh and Turnbull [6] inoculated into one monkey an emulsion of pieces of central nervous system from a fatal case of epidemic encephalitis, and into another monkey a Berkefeld filtrate of this emulsion. The first monkey was killed three weeks later when it was in a drowsy condition. The second monkey died after two months, and the central nervous system showed the typical lesions. No cultural studies with ascitic fluid tissue medium were reported.

MATERIALS

This investigation is based on the following material: [7]

1. The central nervous system was obtained from four cases of epidemic encephalitis, two of the fulminating type, one with marked

4. Flexner, Simon, and Noguchi, Hideyo: J. Exper. M. **28**:461, 1913.

5. Levaditi, C., and Harvier, P.: Compt. rend. Soc. de biol. **83**:354, 1920; Bull. et mém. Soc. méd. d. hôp. de Par. **44**:179 (Feb. 6) 1920.

6. McIntosh, J., and Turnbull, H. M.: Experimental Transmission of Encephalitis Lethargica to a Monkey, Brit. J. Exper. Pathol. **1**:89 (April) 1920.

7. The necropsy material has been secured, through the kindness of Drs. L. M. Warfield and A. G. Margot, from two cases at the Milwaukee County Hospital, and of Drs. A. W. Gray and H. V. Ogden, from cases at Columbia Hospital. The spinal fluids were secured from a patient of Dr. Ogden and from a patient of Dr. Solomon Strouse, at Michael Reese Hospital, Chicago.

myoclonus and one showing the usual form of lethargy. The central nervous system of all had the characteristic lesions described by Marinesco,[8] Bassoe,[9] and others, the changes varying, however, in the different cases. The changes included capillary congestion; microscopic hemorrhages; perivascular, mononuclear leukocytic infiltration; microscopic areas of degeneration and necrosis, etc. The two fulminating cases showed marked congestion, great numbers of capillary hemorrhages, a few areas of focalized necrosis with round cell infiltration and here and there slight or marked perivascular round cell infiltation. The other two cases, which ran a longer clinical course, manifested all these lesions, but many, instead of a few, of the vessels were surrounded by round cells. The degree of this perivascular infiltration was also greater.

2. Spinal fluid was secured from one of the patients with a fatal, fulminating case of epidemic encephalitis and from one patient convalescing from a case of epidemic encephalitis of several months' duration, who had manifested marked lethargy and at the time of lumbar puncture still had headache, nausea, vomiting and a slow pulse.

EXPERIMENTS

About 200 rabbits[10] have been inoculated up to the present time. They received intracranially injections through the thin temporal plate, according to the method devised by Loewe. Young, half grown and full grown rabbits were used. About 1 c.c. can be injected by this method into a full grown rabbit, and it is practically always well tolerated.

To prepare the filtrate, pieces from various portions of fresh central nervous system are ground up in a mortar with sterile sand to make a milky emulsion, and filtered through a Mandler clay filter which has previously been found to hold back *B. prodigiosus*.

At necropsy, on such animals the brain is removed aseptically, most of it is fixed in liquor formaldehydi and cut serially into blocks from which sections are prepared. A large piece is placed in the ice chest in 50 per cent. of glycerol; small pieces are placed in culture tubes. The remainder is ground up with sterile sand, passed through a Mandler

8. Marinesco, G.: Contribution to the Study of the Pathological Histology of Encephalitis Lethargica, Reports to the Local Government Board on Public Health and Medical Subjects, New Ser. 121, 1918, London.

9. Bassoe, Peter: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

10. Recently guinea-pigs have also been used, and a number have developed the experimental disease. These results, together with cultural studies on these guinea-pigs, will be reported later.

filter and the filtrate used for inoculation and cultures. Portions of the cervical and lumbar cord are also placed in liquor formaldehydi.

Of the few animals that died soon after the injection, some died from cerebral hemorrhages, and some apparently from the infectious agent injected. Most of the rabbits died in from two to four weeks after inoculation and some as long as ten weeks later. Some were found dead in the morning after apparently having been well the afternoon before. One apparently well animal, while it was being watched, sprang up in its cage, fell down unable to rise and died in five minutes. Another animal died in the same manner when it was lifted from its cage and placed on the floor for observation. Many animals appeared sick in a general way for a number of days before death, were apathetic and had ruffled fur. Some rabbits developed what seemed to be genuine lethargy. They remained quiet and could not be made to walk. Peripheral paralyses developed in some, involving one or more limbs. One rabbit developed inability to swallow, became emaciated in two days, and was killed. One animal showed myoclonic contractions of the head, eyelids and ears, and also had attacks of excitement culminating in marked opisthotonos, followed by periods of calm.

The animals were inoculated with the same material in groups of from three to eight. In some groups 100 per cent. of the rabbits succumbed, in some as low as 50 per cent. They did not all die after the same interval. At times their deaths were several days or weeks apart. In a number of instances two rabbits in the same group would be found dead the same morning.

Two strains of virus from the brains of two fatal cases have been passed through six series of animals, with filtration through a Mandler clay filter after each passage; that is, the Mandler filtrate from the rabbit brain was injected into the succeeding series of rabbits. The other brain and the spinal fluid strains are more recent and have been passed through only one or two series of animals up to date.

The central nervous system of a large number of the rabbits that died appeared more red and congested than normal, and showed typical and marked microscopic lesions [11] identical with those found in fatal human cases. These lesions were sometimes widespread and sometimes confined to a small portion of the brain or cord. Many sections must be examined so as not to miss the lesions in some animals. Well developed lesions of all the different types found in human cases were at times present in a single rabbit brain. Often, however, the patho-

11. The lesions produced in animals and those present in human necropsy material will be illustrated in subsequent communications.

logic picture was not as complete as this. Microscopic hemorrhages were sometimes the prominent lesion and only an occasional blood vessel showed perivascular round cell infiltration. The brain lesions of some of the rabbits of subsequent series, injected with the same strain of virus showed, however, focalized necroses, characteristic perivascular infiltration, etc. The cord from some animals showed the same lesions. Occasionally only marked congestion was found throughout.

Several series of animals were inoculated with control specimens of spinal fluid and filtrates of brain tissue passed through Mandler clay filters and obtained from cases entirely unrelated to epidemic encephalitis. These animals were chloroformed after varying periods and the brains were found to be normal.

CULTURAL STUDIES

The ascitic fluid tissue culture medium used is the same as that perfected by Noguchi and used by him in the cultivation of *Spirochaeta pallida,* "globoid bodies" of poliomyelitis and other organisms.

Briefly, it consists of a small piece of sterile kidney from a rabbit, ascitic fluid and inoculum. These are placed in a tall, narrow test tube about 1 by 20 cm. About from 10 to 15 c.c. of ascitic fluid [12] are used, making a column from 10 to 15 cm. tall. This is then layered on top with sterile warm petroleum, which, when it cools and hardens, seals the tube. Each material used is controlled for its sterility by the usual bacteriologic method. The tubes are incubated at the usual thermostat temperature, 37.6 C.

Spinal fluid is inoculated directly. Central nervous system material is ground in a sterile mortar, with sterile sand and normal salt solution, and passed through a Mandler clay filter (tested to hold back *B. prodigiosus*). The filtrates thus obtained are usually practically watery clear.

When brain material has been removed aseptically, as from the rabbits, small blocks of the brain are also placed in separate culture tubes.

Smears of the cultures are made by placing a large drop of the culture on a slide with a 1 c.c. pipet; it is then spread with a platinum loop and dried in the incubator. The smears are placed in pure methyl alcohol for 30 minutes and then placed in ether for three minutes. Following the above process the smears are stained in either Loeffler's methylene blue for two hours or overnight in dilute Giemsa (1 part to 20 parts distilled water). Usually in from five to ten days, in the positive cultures, a slight cloudiness appears at the bottom of the tube

12. Dr. A. G. Margot has kindly furnished large amounts of ascitic fluid from the Milwaukee County Hospital.

about the piece of kidney. This may extend upward a few centimeters and only rarely reaches the top. Marked cloudiness, cloudiness of the entire column of ascitic fluid, or cloudiness more marked at the top, usually means an accidental contamination. Accumulation of gas beneath the layer of petrolatum always means a contamination. Contaminations only occur occasionally, which is of interest, since the technic of making these cultures requires that each tube be opened from four to five times. The cloudiness described does not, however, always appear, and the culture may be positive and still remain clear.

The organism stains a violet-blue with methylene blue. With Giemsa, it stains purplish. The organism is extremely minute and rounded (i. e., spherical) and is from one fourth to one fifth the diameter of a small anhemolytic streptococcus. It is found singly, in pairs, in short chains and in groups. It is uniform in size and stands out sharply with the usual magnification of an oil immersion lens. In the stained smears it must be differentiated from amorphous débris from the piece of kidney, from possible granular precipitate from the stain, etc. It is easily differentiated from ordinary contaminating bacteria. Cultures of this organism have been secured from the Mandler filtrates of the central nervous system of the four fatal cases mentioned and directly from the two spinal fluids. Some of these cultures are now in the sixth successful subculture.

Cultures have been obtained from brain blocks and filtrates in about 80 per cent. of the animals successfully inoculated with the original material. The brains of many animals of the different series, inoculated with filtered virus carried through as many as six series of animals, and the brains of animals inoculated with glycerolated virus also yielded positive cultures.

Five rabbits, inoculated with cultures of the first and third generations of several strains of the organism, showed typical brain lesions, and the same organism was recovered from these brains. The organism was also cultivated from Mandler clay filtrates of various positive cultures.

Cultures have been made from time to time of the various types of inocula on the usual laboratory culture mediums. Rosenow cultures in deep tubes of ascitic fluid glucose agar have been made. Except for occasional accidental contaminations with various kinds of organisms, these control cultures have remained sterile.

Control cultures were made in the ascitic fluid—tissue medium of five spinal fluids received in the laboratory for routine examination, one fluid from a case that proved at necropsy to be a brain tumor and one fluid from a case of acute *Staphylococcus aureus* vegetative endocarditis. Mandler filtrates of the brain from this last case, secured at

necropsy, and also of brains from two monkeys that died from generalized tuberculosis were also cultivated. These control cultures did not show the minute oragnism described.

SUMMARY

In the experiments cited it is believed that there has been demonstrated an infectious agent which is apparently constantly associated with epidemic encephalitis. These results are identical with those that have been reported by Loewe and Strauss. They are confirmatory of both their animal experiments and cultural studies. These investigators have, in addition, reported successful experiments on monkeys with the same results as in rabbits.

The infectious agent passes through a clay filter capable of holding back ordinary bacteria (*B. prodigiosus*), the filtrate producing a disease in rabbits which is very similar to the clinical features of the human disease. The microscopic brain lesions are identical with those found in fatal human cases, as described by Marinesco, Bassoe and others. These lesions are the same as those described by Loewe and Strauss and illustrated in their publications.

The virus secured from the original sources, consisting of brains from four fatal cases and from two spinal fluids, has been passed through a number of series of rabbits. Two strains have been passed through six series of animals. The brain emulsion was filtered through a Mandler clay filter after each animal passage. Whatever virus was present in the original material would have been diluted out to an infinitesimal amount before the sixth series of rabbits was reached. This demonstrates the infectious agent, or virus, to be of the class usually termed "filtrable viruses."

By means of ascitic fluid tissue medium, as perfected by Noguchi, an extremely minute filtrable organism has been cultivated from the same infectious material. This organism corresponds exactly in cultural characteristics, morphology, staining characteristics and pathogenic properties to that cultivated by Loewe and Strauss.

This organism has been grown from the brains of about 80 per cent. of the rabbits inoculated with filtered virus. It has also been recovered from the brains of rabbits inoculated with cultures of this organism. Only early cultures, up to the third generation, have been injected. Loewe and Strauss have, however, produced the disease with cultures as far removed as the eleventh generation. They proved, by the following experiment, that the small quantity of original virus was so reduced in amount by subcultures that it could not infect animals, and that the virus (i. e., the filtrable organism) must have proliferated:

Two tenths of a cubic centimeter of a filtered virus, which caused typical lesions in animals and which gave positive cultures of the organism described by them, was placed in 15 c.c. of ascitic fluid, exactly as though making a culture. From this dilution 0.2 c.c. was transferred to a second tube of 15 c.c. of ascitic fluid. This was repeated six times. One cubic centimeter of the sixth dilution failed to infect animals when injected intracranially. This demonstrated that even in six transplantations of cultures the original infectious material was so reduced by dilution that the usual dose of 1 c.c. did not infect animals.

It should be noted that epidemic encephalitis appears with different symptoms, varying greatly in type, location and degree. No two cases present the same clinical picture. Also, the microscopic lesions of the central nervous system show corresponding differences in type, degree and location. Similar variations occur in the disease and in pathologic lesions produced in the animal experiments.

Control studies throughout the investigation were uniformly negative.

CONCLUSIONS

1. The experimental and cultural studies of Loewe and Strauss, on epidemic encephalitis, have subsequently been confirmed by the animal experiments of Levaditi and Harvier, McIntosh and Turnbull, and by the experimental and cultural studies reported in this paper.

2. The extremely minute filtrable micro-organism, that has been successfully cultivated in ascitic fluid tissue medium from brain material and spinal fluid obtained from patients with epidemic encephalitis, is believed to be identical with the micro-organism that has been cultivated and reported by Loewe and Strauss.

3. The investigations of Loewe and Strauss, followed by those of Levaditi and Harvier and of McIntosh and Turnbull, taken together with those reported by us, strongly indicate that epidemic encephalitis is caused by a filtrable virus.

4. The filtrable micro-organism cultivated by Loewe and Strauss, and also subsequently cultivated in the course of these studies, is believed to be the etiologic agent of epidemic encephalitis.

JUVENILE TABES

REVIEW OF THE LITERATURE AND SUMMARY OF SEVEN CASES *

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LITERATURE ON JUVENILE TABES

It is natural that the very existence of juvenile tabes dorsalis should be questioned. Among those doubting the reality of such a disease were Marie,[1] von Leyden [2] and Gumpertz.[3] Yet it has now come to be accepted, and the increase in knowledge brought by the Wassermann test and the procedure of spinal puncture has not merely increased the facility of its recognition, but has also widened the scope of our knowledge of its manifestations.

Remak,[4] in 1885, described the first case of juvenile tabes. Since that time many cases have been reported. On account of the varying standards of what comprises a disease, the literature, up to the time of the recognition of syphilis as a causative factor and of the Wassermann test, was very contradictory. From time to time leading writers have collected series of cases and analyzed them, selecting what in their judgment constituted the picture of juvenile tabes, and rejecting in some cases the very cases on which previous authors had laid greatest stress. Today the literature on the subject may be divided into analyses of collected series and reports of isolated cases. I shall try to give a comprehensive review of the collected series and an analysis of ten isolated cases that I have collected.

Juvenile tabes is a rare disease and quite uncommon as compared with the diffuse form of neurosyphilis of children. It is rare also as compared with juvenile paresis. There are probably ten cases of juvenile paresis to one of juvenile tabes (Jones [5]), and, although adult tabes is more common than adult paresis, the reverse is true in the

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*Work done in the Section on Neurology, Mayo Clinic.

1. Marie, P.: Leçons sur les maladies de la moelle, Paris, G. Masson, 1892.
2. Leyden, E. von: Die Tabes dorsalis, Real-Encycl. d. ges. Heilk. **24**:68, 1900.
3. Gumpertz, K.: Was beweisen tabische Symptome bei hereditär syphilitischen Kindern für die Aetiologie der Tabes? Neurol. Centralbl. **19**:803, 1900.
4. Remak, B.: Drei Fälle von Tabes im Kindesalter, Berl. klin. Wchnschr. **22**:105, 1885.
5. Jones, E.: The Symptoms and Diagnosis of Juvenile Tabes, Brit. J. Child. Dis. **5**:131, 1908.

juvenile forms. In spite of its rarity, however, we no longer doubt that there is a definite disease called juvenile tabes, which may produce any one or every one of the signs and symptoms of adult tabes.

There seems to be no great predilection for either sex in the juvenile form, whereas in the adult form the predatory male more often acquires syphilis and later tabes. Females seem slightly to predominate in the juvenile forms; in three series there were fifty-eight girls to forty-five boys. In Cantonnet's [6] series 62 per cent. were girls and 37 per cent. boys.

With regard to the age incidence, account must be taken as to whether the syphilis is congenital or acquired. In the acquired cases, it is easy to understand that syphilis must be acquired early to produce juvenile tabes. In a case Marburg [7] records, the child was infected at the age of 2 by a nurse and developed tabes at 8, the youngest patient with tabes due to acquired syphilis on record. In the cases of congenital syphilis, by far the greatest number in the various series, the age incidence was as follows:

In the Hirtz and Lemaire [8] series the average age was 14.3; in Cantonnet's series the average age was 15, and in the series of ten cases that I collected the average was 14. The average age is, therefore, about 14, or puberty. The youngest of the patients was one of Mingazzini and Baschieri Salvedori.[9] The child was 3 years old when symptoms began. The youngest in Hirtz and Lemaire's series was 6. Bertolotti [10] reported a case of tabes due to inherited syphilis in which the patient was 40. That syphilis is the chief factor in the etiology of the disease is now accepted, but to trace its origin in some of the cases is interesting, considering how much in doubt the older writers were as to the part it played in the disease. With our modern methods for the detection of the disease, we do not lay so much stress on the absence or the presence of a history of parental syphilis as did early authors. The parental origin of infantile syphilis is now regarded as demonstrated when the whole family gives a total inhibition Wassermann test, although the history reveals no trace of acquired syphilis in the parents. We may suspect that it is a case of acquired juvenile

6. Cantonnet, A.: Les manifestations oculaires du tabes juvenile, Arch. d'ophth. **27**:708, 1907.

7. Marburg, O.: Klinische Beiträge zur Neurologie des Auges. Infantile und juvenile Tabes, Wien. klin. Wchnschr. **16**:1295, 1903.

8. Hirtz, E., and Lemaire, H.: Etude critique sur le tabes infantile juvenile, Rev. neurol. **13**:265, 1905.

9. Mingazzini, G., and Baschieri-Salvadori, G.: Considerazioni cliniche sulla tabe ereditaria, Riv. di patol. nerv. **11**:580, 1906.

10. Bertolotti, M.: Tabe ereditaria tardiva per sifilide congenita, Riforma med. **21**:93, 1905.

tabes when the tests of both parents fail to show evidence of syphilis. In a case in the records of the Mayo Clinic a child had all the signs of juvenile tabes, but her supposed mother, who was being examined at the same time, had no signs of syphilis, including a negative Wassermann test. The child proved, after some inquiry, to be an adopted daughter of doubtful parentage. Nonne [11] reports a child infected at the age of 5 through sleeping with a lodger. The child conveyed the disease to both parents by kissing; finally all three developed tabes. Nonne also tells of a mother who infected herself wet nursing an infant, and then infected her own child, who later developed juvenile tabes.

The number of recorded cases of juvenile tabes due to acquired syphilis is relatively small compared with the number due to hereditary syphilis. In Lasarew's [12] twenty-three cases only two were recorded as due to acquired syphilis, and in Hirtz and Lemaire's series of forty-six cases of juvenile tabes syphilis was acquired early in childhood in only three. A patient to become tabetic while still juvenile, that is under 21, must necessarily receive the primary lesion at an early age. Von Halban [13] recorded a case in which the child at 4 months of age was infected with syphilis from a nurse; later it developed juvenile tabes. Kutner [14] described a case in which the child was infected at 5 by the kiss of a prostitute.

The question of specificity of spirochetes that produce types of diseases of the central nervous system as compared with the influence of the soil they settle on enters into this disease. Is there a particular strain of spirochetes which produces tabes or general paresis in a person, or do the results of infection depend on the type of his nervous system or his family predisposition, or, in short, on some condition for which he is responsible irrespective of the strain of the spirochetes? This is partially answered by the characteristics of juvenile tabes. The frequency with which the parents of juvenile tabetic patients are either paretic or tabetic stands out. Cantonnet, in his series, showed that in 15 per cent. one parent was tabetic and in 12 per cent. paretic.

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11. Nonne, M.: Syphilis und Nervensystem, Berlin, Karger, 1902; Ueber die Bedeutung der Syphilis in der Aetiologie der Tabes, Fortschr. d. Med., No. **29**:986, 1903; Ein Fall von familiärer Tabes dorsalis auf syphilitischer Basis. Tabes bei der Mutter und ihren zwei hereditär-syphilitischen Töchtern, Berl. klin. Wchnschr. **41**:845, 1904.

12. Lasarew, W.: Ein Beitrag zur Tabes in jungem Alter (Tabes infantilis und juvenilis), Neurol. Centralbl. **24**:988, 1905.

13. Halban, H. von: Ueber juvenile Tabes nebst Bemerkungen über symptomatische Migraine, Jahrb. f. Psychat. u. Neurol. **20**:343, 1901.

14. Kutner, R.: Ueber juvenile und hereditäre Tabes dorsalis, Inauguration Dissertation, Breslau, 1900.

Nonne records a case in which a sister, as well as the mother, had tabes, also the case of a brother and sister with tabes, and an instance of twin brothers developing tabes at the same age. Grinker [15] reports a family in which the father was syphilitic, the mother tabetic, one son paretic and a daughter hemiplegic. Smith [16] also reported a family of four; the father had paresis, the mother Argyll Robertson pupils, one son juvenile paresis, and one daughter juvenile tabes. Since these few cases were reported many more have been recorded, showing how frequently a family of four or five becomes affected with the same type of neurosyphilis. In some cases a definite neuropathic family tendency can be demonstrated which seems to be a suitable soil for the production of tabes or paresis. On the other hand, it has been shown again and again that paresis or tabes, or both, may be produced in several persons by infection from the same source. The question of the relative importance of a neuropathic heredity and of a possible specific syphilitic nerve virus is yet to be settled.

The course of juvenile tabes is always long and protracted. Juvenile tabes is never acute or fulminating, as adult tabes is occasionally. All observers who have had the opportunity of following the cases for long periods agree to this. Mendel,[17] in 1895, described a case a report of which was first published by Remak in 1885, then by Hildebrand [18] in 1892. In these ten years the progress of the disease was exceedingly slight. Two things follow from this chronicicity: First, cases in adults are likely to be diagnosed as acquired tabes unless a careful history is taken; and second, necropsies in these cases are few because in the lengthy course of the disease the patients drift away from observation. Cantonnet followed the histories of twelve patients from ten to twenty years; there was not a death during that time. Large numbers of juvenile tabetic patients, however, drift into taboparesis; even larger numbers become paretic and asylum inmates, and the final change clouds the initial tabes. The rare recognition of juvenile tabes is partly explained by the later change.

No symptom that occurs in adult tabes may not occur in the juvenile form (Skala [19]). The symptoms predominate in a different manner, however; some are more common in juvenile than in adult tabes, and

15. Grinker, J.: A Case of Juvenile Tabes in a Family of Neurosyphilitics, J. Nerv. & Ment. Dis. **31**:753, 1904.

16: Smith, H. J.: Presentation of a Tabetic Child and Paretic Parents and Brother, J. Nerv. & Ment. Dis. **44**:66, 1916.

17. Mendel, E.: Die hereditäre Syphilis in ihren Beziehungen zur Entwicklung von Krankheiten des Nervensystems, Festschr. f. G. Lewin, Beitr. z. Dermat. u. Syph. 1896, p. 138.

18. Hildebrand: Quoted by Jones.

19. Skala: Quoted by Jones.

some of the commoner symptoms in the adult disease are rarely seen in the juvenile. Vesical disturbance is most often incontinence, either diurnal or nocturnal, in the latter case not to be distinguished from the common bed wetting of infants. In the order of frequency of symptoms, it is the most constant and earliest and occurred in nearly half of the cases in Hirtz and Lemaire's series and was initial in twelve twenty-eighths. Dydynski [20] gave it equal importance with optic atrophy. Marburg found it in twenty of thirty-four of his cases, and Lasarew in fourteen of twenty-three. Lasarew found it in the initial symptom in seven of twenty-three. In the series of isolated cases which I collected, it occurred in five of ten, 50 per cent. The average percentage of all series is 55.

Ocular symptoms follow incontinence in frequency, and in some series equal vesical disturbances in their constancy and frequency as initial symptoms. The first symptom was amblyopia due to optic atrophy in 14 per cent. of Cantonnet's cases. The same percentage occurred in Hirtz and Lemaire's series. As a definite finding it was present as follows: Cantonnet's series, 43 per cent. of 89 patients; Hirtz and Lemaire's series, 36 per cent. of 46 patients; Marburg's series, 33 per cent. of 34 patients; collected cases, 70 per cent. of 10 patients; average, 45 per cent. Other ocular findings, such as Argyll Robertson pupils were present in 87 per cent. of Lasarew's cases, in 73 per cent. of Marburgs, in 73 per cent. of Hirtz and Lemaire's and in 80 per cent. of the series I collected; the average is 78 per cent. This is practically the same percentage as in adult tabes. Irregularity and inequality of the pupils occur with about the same frequency. Ocular palsies and diplopia, if they occur at all, are late symptoms (Jones).

Lightning pain was the initial symptom in 25 per cent. in Cantonnet's series, and in Hirtz and Lemaire's series. They were present in Lasarew's series in 91 per cent., in Marburg's series in 71 per cent., in Hirtz and Lemaire's series in 32 per cent., and in my collected cases in 10 per cent. The average percentage was 51. Jones believes that after explaining the discrepancy by the difference of opinions of authors as to which cases shall be termed juvenile tabes, the number of patients affected by lightning pains is about the same in both types of tabes, that is, 88.4 per cent.

Headache has been included by Jones as a common symptom. Von Halban laid great stress on the appearance of migraine early in the disease. Girdle pains are rarer in the juvenile form than in the adult

20. Dydynski, L. von: Tabes dorsalis bei Kindern, nebst einigen Bermerkungen über Tabes auf der Basis der Syphilis hereditaria, Neurol. Centralbl. **19**:298, 1900.

form. They occur in only 16 per cent. of the total number of Lasarew's and Marburg's cases.

Sensory changes were present in 65 per cent. of Hirtz and Lemaire's series, in 91 per cent. of Lasarew's cases, in 91 per cent. of Marburg's cases, and in 30 per cent. of the cases I collected, a fairly large discrepancy between the series, which gives an average of 68 per cent. that must be accepted with caution.

All writers agree that the most striking phenomenon in the adult form of the disease, the ataxia which Duchenne so carefully described, is relatively rare in juvenile tabes. Marburg estimates it as occurring in 33 per cent. of the cases, Linser,[21] in 33 per cent., Lasarew in 63 per cent. and Hirtz and Lemaire in 4 per cent.; in my series it occurred in 20 per cent. This gives an average of 30 per cent. and contrasts remarkably with the series in the adult type in which the frequency has been estimated as 66.6 per cent. Roughly, only one third of juvenile tabetic patients are ataxic, while twice that number of adult tabetic patients have difficulty in locomotion. Cantonnet states that the difficulty is unusual, and he explains it as being due to optic atrophy. Dydynski states that ataxia is either absent or late in appearing in juvenile tabes. The fact that this symptom is rare does not preclude the possibility of its being present and present early (Jones).

Gastric crises are not unknown in juvenile tabes; they are not even rare; 17 per cent. of the patients of Lasarew's series complained of them, 20 per cent. of Marburg's, and 19 per cent. of Hirtz and Lemaire's, giving an average of 18 per cent. thus affected.

Trophic disturbances have been recorded. Nonne has described a case in which a juvenile tabetic patient developed a Charcot joint. Romberg's sign was seen in 75 per cent. of the series analyzed and Westphal's sign was found in about 90 per cent. In the series I collected the patellar reflexes were absent in 80 per cent.

From the foregoing survey of these series of cases of juvenile tabes, we are now in a position to draw some conclusions with regard to the relative frequency of the different symptoms and their order of appearance, and to compare them with the symptoms in adult tabes. Most remarkable is the frequency of vesical disturbance and amblyopia, both as initial symptoms and as constant features of the disease. Hirtz and Lemaire call attention to the insidious manner in which the disease appears, how often the oculist, after discovering early optic atrophy in a child thought of testing the patellar reflexes and was first to diagnose a tabes that had not been ushered in by such striking symptoms as ataxia or lightning pains. In 16.66 per cent. of the Hirtz-Lemaire

21. Linser, P.: Ueber juvenile Tabes und ihre Beziehungen zur hereditären Syphilis, München. med. Wchnschr. 1:637, 1903.

series incontinence and slight sensory changes were the only symptoms calling attention to the disease, and it was only after finding Westphal's sign and Argyll Robertson's pupils that a suspicion arose as to the nature of the disease.

So much for the gleanings from literature up to 1908, when our knowledge of neurosyphilis was not so complete as it is now. In those days when diagnosis was made chiefly by symptoms and physical signs, so insidious a disease as juvenile tabes was overlooked first, because its relation to syphilis was still viewed with suspicion, and second, because its syphilitic origin granted, there was no Wassermann test to fill in the gaps left by the absence of stigmas or family history of the disease. Just because so striking a sign as ataxia was rare, its existence was doubted, and worse still, those who accepted it as an entity were ready to confound it with Friedreich's ataxia and cerebellar ataxia and to write articles on cases they imagined were types of the disease. Whereas in the latter part of the twentieth century ataxia was the deciding point in the diagnosis, now a definite finding of syphilis is demonstrated. The general accumulation of data has shown the existence of the disease, shown the differences, and raised the question as to why there should be differences. I believe that an infection implanted on a tissue that has not yet fully developed ought to produce a different result from that obtained when the same infection is sown on a fully differentiated and completed nervous system. In the selection of the cases of juvenile tabes from the records of the Mayo Clinic, I have guided myself by giving most importance to (1) definite evidence of syphilis, (2) presence of Argyll Robertson pupils, (3) optic atrophy, (4) Westphal's sign, or absent tendo-achillis reflexes, (5) incontinence of urine, and (6) absence of all mental deterioration. The case will be described in the order of the patients' admission to the Clinic.

REPORT OF CASES

Case A151370.—R. M., a young man, aged 17, came to the Clinic Feb. 3, 1916, complaining of vomiting that was without any relation to meals. He gave a typical history of gastric crises, beginning at the age of 11. Examination showed a striking condition of hypotonia and such laxity of the joints that the patient could twist his body and limbs into most grotesque postures. There were Hutchinson's teeth, a saddle shaped nose, external strabismus, and unequal pupils; Argyll Robertson pupils, Westphal's sign, no Achillis' tendon reflex, and some diminution of pain and temperature sensation over the forehead and feet. No Romberg sign, ataxia, or incoordination was present. The Wassermann test on the blood was strongly positive. Examination of the spinal fluid was negative.

Case 205051.—E. S., a boy, aged 7, was brought to the Clinic Aug. 15, 1917. For one year before examination the child had had frequent attacks of vomiting, lasting from three to six days, and ending suddenly. He felt well between

attacks and ate to make up for the loss during attacks. He urinated involuntarily and had difficulty in starting the stream. Examination showed general adenopathy and carious teeth. The pupils did not react to light or during accommodation; Westphal's sign was present, the tendo-achillis reflex was diminished, and sensation of pain was diminished over the third dorsal segment. Abadie's sign and some hypotonia were present; there was no Romberg's sign, ataxia, or incoordination. The Wassermann test on the blood was positive, also in the father and mother.

Case 241224.—K. H., a boy, aged 14, came to the Clinic Aug. 6, 1918, complaining of blindness, which had begun four years before; at the time of examination he was blind in the left eye and could see only 2/200 in the right eye. He had had lightning pains during the last two years. Examination showed an early saddle nose and Hutchinson's teeth. The left pupil was larger than the right, and both were irregular. Reflexes to light and during accommodation were absent. There were complete primary optic atrophy of both nerve heads, hypotonia of all the muscles, and diminished sensation of pressure and vibration over both lower extremities. Westphal's sign and diminished tendo-achillis reflex were present. Examination of the spinal fluid showed a strongly positive Wassermann reaction with 0.3 and 0.4 c.c., a positive Nonne reaction, and 83 small lymphocytes. No Lange test was performed. He gave a strongly positive Wassermann reaction on the blood.

Case 303391.—C. V. S., a boy, aged 15, presented himself Jan. 20, 1920, complaining of blindness. Seven years before he could see well; since that time sight had been failing gradually, and he was able to distinguish only light and dark. His pupils reacted neither to light nor during accommodation; there were simple optic atrophy of the right eye and an interstitial keratitis of the left. Examination showed a strongly positive total inhibition Wassermann test of his blood. Westphal's sign was present, and the tendo-achillis reflexes were absent. Some slight incoordination was found, and Romberg's sign was present. There was diminution of pain sensibility over the arms and legs. Vibration sensibility was diminished in the lower extremities. The spinal fluid examination showed a negative Wassermann and Nonne test and 36 small lymphocytes.

Case 305100.—P. F., a young woman, aged 19, presented herself for examination Feb. 4, 1920, complaining of blindness and headaches. She had had frontal headaches since the age of 12, and failing vision in both eyes for the last four years until she barely had light perception. The left eye failed first. Her gait had been staggering and uncertain, and for the last eighteen months she had had pains in the calves corresponding to lightning pains. She had received five intracranial arsphenamin injections through a trephine hole in the skull; but there had been no improvement. Examination showed simple primary optic atrophy complete in both optic disks. Pupil reflexes were absent. There was a diminution in the vibration sense in both upper and lower extremities. Joint sensibility was almost absent in both feet. Westphal's sign was present and the tendo-achillis reflexes were absent. There was great incoordination in the heel and knee test; the gait was extremely ataxic and the Romberg sign was positive. The Wassermann test of the blood was negative, but the spinal fluid showed a total inhibition Wassermann with 0.3 and 0.4 c.c., a negative Nonne reaction, 306 lymphocytes and 32 polymorphonuclears.

Case 306325.—N. N., a young woman, aged 19, presented herself for examination Feb. 16, 1920, complaining of pain in the neck, shoulder and occiput, which

had commenced fifteen years before, frequent and severe sore throat and insomnia. Examination showed convergent strabismus of the right eye, irregular and Argyll Robertson pupils. There was slight incoordination of the hands, Westphal's sign was present and the tendo-achillis reflex was absent. Vibration sense over both malleoli was diminished; there was no Romberg sign, no ataxia, no incoordination. The Wassermann test of the blood serum was strongly positive with total inhibition, while the Wassermann test with 0.3 and 0.4 c.c. of the spinal fluid was strongly positive also with total inhibition. There was a negative Nonne reaction, 240 small lymphocytes, and a "syphilitic curve" of 1112221000 in the Lange test.

Case 266511.—G. B., a boy, aged 12, was admitted to the hospital April 3, 1919, complaining of pains in the joints of the wrists, elbows and knees, and of attacks of abdominal pain; the joints were swollen and tender. At times the patient could not hold his urine. Examination showed no hereditary stigmas, but the blood gave a positive Wassermann test with total inhibition. One pupil was much larger than the other and both were irregular. Argyll Robertson's pupils and Westphal's sign were present. Both tendo-achillis reflexes were absent, but the Romberg test was negative. There was no ataxia or incoordination. The boy was seen a year later, but, although examination showed a complete cessation of joint trouble, all other findings were the same; he complained of shooting pains on the outer side of his legs. The spinal fluid was negative.

CONCLUSIONS

To draw any sweeping conclusions from so small a number of cases would invite error; however, something may be learned from their study.

In all seven cases Westphal's sign was present, in six some degree of diminution of sensibility was found, in four the pupils were immobile, in three Argyll Robertson pupils were present, in three optic atrophy, in three crises, in three lightning pains, in three incoordination, in three signs of congenital syphilis, or syphilis outside the central nervous system, in three hypotonia, and in two incontinence of urine. In one only was ataxia present. The serum Wassermann test was triple positive in all but one case. In six cases a spinal fluid examination was made. In half that number the Wassermann test was negative. One of three cases in which the spinal fluid Wassermann test was negative had a cell count of 36. A Lange test was performed once, only, and that had a "syphilitic curve." The patient who gave a negative serum Wassermann reaction also gave a total inhibition spinal fluid Wassermann reaction. Without drawing conclusions, it is possible to remark on (1) the frequency of some pupillary change, (2) the frequency of sensory changes, (3) the equal frequency of optic atrophy, crises, lightning pains, and incoordination, and (4) the relative infrequency with which incontinence of urine and ataxia were encountered, especially ataxia. It seems remarkable that in the six spinal fluids examined, three should have been negative in spite of well-marked symptoms and positive blood serum.

Hypotonia, a phenomenon not mentioned in any of the literature I read, was present in three cases, and in one case was the most striking feature. At the same time, children are much more flexible than adults, and how much is normal or how much abnormal for a child is hard to determine.

Another noticeable feature is that of seven patients with disturbances of pupil reflexes; four had no movement to light or during accommodation. The large immobile irregular pupil is more frequent with juvenile tabetic patients in contrast to the myosis and Argyll Robertson pupil of adult patients.

In the comparison of my findings in these few cases with those of former authors there is little essential difference, except that I found vesical incontinence not so frequent as reported. Perhaps if a larger number of cases had been at hand, my percentage would have been larger. The cases agree well with regard to the paucity of physical signs, the long insidious course and the rarity of ataxia. The Wassermann tests of the serum and spinal fluid have simply made the diagnosis easier.

SUMMARY

Juvenile tabes is now a well recognized entity, and with our increased facility of diagnosis, more and more cases are being recognized and reported. The features of the disease that I have attempted to show may be briefly summarized: The insidious onset, the lengthy and even latent course of the disease, during which time no subjective complaint may be made, are in contrast to the frequent optic atrophy and total blindness that may also occur. The frequency of incontinence of urine, the relative rarity of such striking phenomena as ataxia, girdle sensations and lightning pain, and finally the frequent paretic termination, are features that stamp juvenile tabes with a distinctive mark and distinguish it from the adult type.

INTERPRETATION OF THE "PARETIC CURVE" IN LANGE'S COLLOIDAL GOLD TEST*

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The occasional occurrence of a paretic gold curve in cases that were not paresis, and the occasional diagnosis of paresis without a paretic gold curve suggested that a closer study of this particular colloidal gold reaction should be made. A review of the rather extensive literature that has grown up since Lange[1] first introduced this test in 1912 shows that various conclusions have been drawn concerning the value of the paretic curve. These conclusions, however, were based on a more or less limited number of cases.

At this hospital about 8,400 colloidal gold tests have been made on about 7,100 different cerebrospinal fluids. Of this number, 677 fluids gave paretic curves, and these constitute the basis for this study. A large majority of the patients coming to this hospital are in the early stages of their disease, and consequently these findings are those of the early stages, and are, therefore, the more valuable. Another advantage is that in almost every instance a fluid is obtained before antisyphilitic treatment is started. The diagnoses of all the cases considered have been confirmed by a long period of observation at this or some other state hospital, and in addition many have been confirmed by necropsy.

The preparation of the colloidal gold, the technic of the test, and the reading of the results have all been adequately described by many writers;[2] these points will, therefore, not be taken up in this article. I shall consider only the interpretation of the paretic gold curve.

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* From the Massachusetts State Psychiatric Institute, Boston.

1. Lange: Ueber die Ausflockung von Gold Sol durch Liquor Cerebrospinalis, Berl. klin. Wchnschr. **49**:897, 1912,

2. Lange: Ueber die Ausflockung von Gold Sol durch Liquor Cerebrospinalis, Berl. klin. Wchnschr. **49**:897, 1912. Kaplan: Serology of Nervous and Mental Diseases, Philadelphia, W. B. Saunders Company, 1914. Miller, Brush, Hammes and Felton: A Further Study of the Diagnostic Value of the Colloidal Gold Reaction Together with a Method for the Preparation of the Reagent, Bull. Johns Hopkins Hosp. **26**:391, 1915. Warwick and Nixon: A Study of the Colloidal Gold Reaction and Its Clinical Interpretation, Arch. Int. Med. **25**:119 (Feb.) 1920. Lowrey: Cerebrospinal Fluids, Especially the Gold Reaction in Psychiatric Diagnosis, J. Nerv. & Ment. Dis. **46**:186 (Sept.) 1917.

First, what shall be called a paretic curve? It is agreed that the typical paretic curve gives the following reading: 5555543100,[3] but there are many variations that may still be called paretic curves. The number of tubes showing complete precipitation may be increased or decreased somewhat without altering the interpretation. In one case of undoubted paresis after an acute exacerbation and after some intraspinal treatment, all the usual ten tubes were reduced to fives with a sudden drop to the normal color in further dilutions. This case had previously given typical paretic curves, and soon after the typical curve reappeared. It also seems from experience with other cases that the number of fives may be reduced to two or three and the result may still be called a paretic curve if the general form of the typical curve is present. A reading such as 4444432100 should certainly be called a paretic curve, especially when the fours are well marked. However, if the reading starts with only threes, we are on the borderline between the paretic and the syphilitic curve. Therefore, if the general conformation of the typical paretic curve is present, and the reduction in the first three or more tubes is well marked in the fours or is in the fives, we may consider it a paretic curve. An irregular reading, such as 4525231000, may be found after intensive treatment, as a result of poor gold solution or in contaminated fluids, and such a reading should always be questioned.

GENERAL CONSIDERATIONS

Because of the name, "paretic curve," the question is often asked, "Does a paretic curve always mean paresis?" or in other words, "Is it pathognomonic of paresis?" This question has been answered in the negative by many previous writers. Of this series of 677 paretic curves, thirty-nine cases were not paretic; of these fourteen were not cases of neurosyphilis (Table 1).

PARETIC CURVES IN PARESIS

Another question often asked is whether or not the fluids from cases of paresis always give paretic curves. This can also be answered in the negative by the fact that of 638 cases of paresis, a paretic curve was not found in the first fluid tested in eleven cases. However, four of these later developed a typical curve, and in the remainder only one specimen of spinal fluid was examined.

Among the cases of paresis just mentioned are included the cases diagnosed as taboparesis and juvenile paresis. These types gave typical results in every instance but two. A few other cases were found in

3. Solomon, Koefod and Welles: The Diagnostic Value of Lange's Gold Sol Test, Boston M. & S. J. 173:957, 1915.

TABLE 1.—General Distribution of Paretic Curves

Author	Number of Paretic Curves	In Paresis		Cerebrospinal Syphilis or Tabes		In Diseases Other Than Neurosyphilis	
		Number	Percentage	Number	Percentage	Number	Percentage
Miller, Brush, Hammes and Felton[4]	151	129	85.4	19	12.5	3	2.1
Lowrey [5]	69	65	93.6	3	4.3	1	2.1
Swalm and Mann [6]	62	62	100.0	0	0.0	0	0.0
Rawlings [7]	102	101	99.0	0	0.0	1	1.0
Hammes [8]	52	42	80.7	9	17.3	1	2.0
Eicke [9]	50	50	100.0	0	0.0	0	0.0
Jaeger and Goldstein [10]	48	33	68.7	12	25.0	3	6.3
Eskuchen [11]	18	17	94.4	1	5.6	0	0.0
Cases in this series	677	638	94.2	25	3.7	14	2.1

4. Miller, Brush, Hammes and Felton (Footnote 2).

5. Lowrey (Footnote 2).

6. Swalm and Mann: New York M. J. **101**:719, 1915.

7. Rawlings: The Colloidal Gold Reaction in Four Hundred and Ninety-Eight Psychiatric Cases, Arch. Neurol. & Psychiat. **2**:180 (Aug.) 1919.

8. Hammes: The Comparative Value of the Wassermann, the Colloidal Gold and Other Spinal Fluid Tests, Am. J. M. Sc. **154**:625 (Nov.) 1917.

9. Eicke: Die gold reaktion im liquor cerebrospinalis, München. med. Wchnschr. **60**:2713, 1913.

10. Jaeger and Goldstein: Goldsolreaktion im liquor cerebrospinalis, Ztschr. f. d. ges. Neurol. u. Psychiat. O. **16**:219, 1913.

11. Eskuchen: Die fünfte reaktion, Ztschr. f. d. ges. Neurol. & Psychiat. O. **25**:486, 1914.

TABLE 2.—Cases of Paresis Without a Paretic Curve in the First Fluid Tested

Number	Date	Colloidal Gold Curve	Glucose	Albumin	Cell	Wassermann Test		Remarks
						Cerebrospinal Fluid	Serum	
6941	6/24/16	0001232100	3	3	58	55000*	Positive	Taboparesis. On admission showed typical symptoms of paresis
	7/25/16	5555533000	3	3	24	55500	Positive	
11514	10/29/18	2223310000	0	N	0	Positive	Negative	Diagnosis of paresis recently confirmed by necropsy
	11/12/18	5554443310	0	1	1	Positive	Positive	
4303	11/17/15	3333210000	2	2	55	Doubtful	Positive	Typical paretic symptoms
9217	8/16/16	3333211000	1	1	27	Positive	Positive	Cause of death, general paralysis
12036	1/27/19	0004521000	1	1	2	Positive	Positive	Cause of death, general paralysis
11123	8/10/18	1112321000	2	2	116	50000	Positive	Diagnosis, taboparesis; treated since first L.P.
	12/ 2/18	5554443221	1	1	2	555540	Positive	
	11/ 2/19	5555443100	1	1	2	555550	Positive	
9496	10/14/17	3332100000	3	3	27	Positive	Positive	Diagnosis confirmed at Taunton State Hosp.
11981	1/17/19	0001131100	1	2	17	Positive	Positive	Diagnosis confirmed at Westboro State Hosp.
9244	8/21/17	3333210000	1	1	46	Positive	Positive	Diagnosis confirmed at Taunton State Hosp.
8520	4/18/17	1132210000	0	2	38	Positive	Positive	Typical paretic
7788	12/ 6/16	2221000000	3	4	59	55555	Positive	Treated after first L.P.
	12/23/16	5555443300	3	3	40	50000	Positive	
	1/24/17	1112310000	3	4	23	55555	Positive	

* The reaction is recorded in various dilutions of spinal fluid from 2 c.c. to 0.1 c.c. The figure 5 means a strongly positive reaction.

which there was not a paretic type of curve, but all of the patients had received previous intensive treatment before they were examined at this hospital.

The objection may be made that few cases were diagnosed as paresis in the absence of a paretic curve. That is the tendency, but it was found that many cases were diagnosed as paresis without a paretic curve; most of them, however, were later proved to be incorrectly diagnosed. One case, for instance, was consistently diagnosed as general paralysis in spite of the absence of a paretic curve in several fluids tested, but at necropsy no evidence, either microscopic or macroscopic, could be found to support this diagnosis.

TABLE 3.—PARETIC CURVES IN PARESIS

Author	Number of Cases	Colloidal Gold Curve								Wassermann Test	
		Paretic		Syphilitic		Atypical		Negative			
		Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage	Percentage Positive	Percentage Negative or Doubtful
Lowry [5]	94	69	73.4	7	7.4	16	17.0	2	2.2	88.3	9.7
Eskuchen [11]	21	17	81.0	3	14.0	1	5.0	0	0.0	62.0	38.0
Hammes [8]	43	42	98.0	1	2.0	0	0.0	0	0.0	96.0	4.0
Eleks [9]	52	50	96.0	0	0.0	0	0.0	2	4.0	96.0	4.0
Larkin and Cornwall [12]	99	86	87.0	—	—	—	—	—	—	95.0	5.0
Rawlings [7]	120	101	84.0	7	6.0	11	9.0	1	1.0	96.0	4 0
Swalm and Mann [6]	70	62	89.0	3	4.0	4	5.7	1	1.3	92.8	7 2
Miller and Levy [13]	49	49	100.0	0	0.0	0	0.0	0	0 0		
Miller, Brush, Hammes and Felton [4]	130	126	96.9	1	1.0	2	2.0	2	2.0	93.0	7.0
Weston [14]	34	31	91.0	3	9.0	0	0.0	0	0.0	91.0	9.0
Jaeger and Goldstein [10]	33	33	100.0	0	0.0	0	0.0	0	0.0	94.0	6.0
Present series	638	627	98.2	8	1.2	3	0.6	0	0.0	96.4	3.6

12. Larkin and Cornwall: Value of Laboratory Diagnosis in Neurosyphilis, Am. J. Syphilis **3**:76, 1919.

13. Miller and Levy: Colloidal Gold Reactions in the Cerebrospinal Fluid Bull. Johns Hopkins Hosp. **25**:133, 1915.

14. Weston: Colloidal Gold and Other Tests Applied to the Spinal Fluid, Am. J. Insan. **71**:773, 1914.

PARETIC CURVES IN OTHER DISEASES

Cerebrospinal or Diffuse Neurosyphilis.—The differentiation of cerebrospinal syphilis from paresis is difficult, especially in the early stages, and apparently the colloidal gold test is not to be relied on entirely when such a question arises. In cerebrospinal syphilis the syphilitic type of curve (1233210000 or 3332110000) is most often found. However, in the series of cerebrospinal syphilis cases at this hospital several paretic curves were found in the first fluid examined, and some fluids developed a paretic reaction later. No case of this type was considered unless it was so typical that paresis could be

excluded entirely. Of thirty-one cases of cerebrospinal syphilis, fifteen gave a paretic curve in the first fluid tested and six gave syphilitic curves; in four the colloidal gold curve was negative, and the remaining six gave atypical readings. Of the fifteen cases with paretic curves, all but three showed marked psychotic symptoms, while those with the milder reaction showed chiefly physic symptoms, such as cranial nerve palsies, and the mental symptoms were more or less in the background or entirely absent. In this type of neurosyphilis there may be parenchymatous involvement or simply meningitic and vascular changes. It would seem, therefore, from these findings that the paretic curve in cerebrospinal syphilis would indicate parenchymatous involvement.

TABLE 4.—Colloidal Gold Tests in Cerebrospinal Syphilis

Author	Number of Cases	Paretic Curves	Syphilitic Curves	Chronic Type	Atypical Curves	Negative
Warwick and Nixon [15]	26	3	10	—	9	4
Hammes [8]	27	3	23	..	1	0
Black [16]	39	1	15	20	—	3
Rawlings [7]	59	1	33	..	14	3
Swalm and Mann [6]	10	0	6	—	—	4
Miller, Brush, Hammes and Felton [4]	15	4	9	—	—	2
Eskuchen [11]	13	1	12	—	—	—
Eicke [9]	25	0	24	—	—	1
Cases in this series	31	15	6	—	6	4

15. Warwick and Nixon (Footnote 2).

16. Black, J. H.; Rosenberg, L., and McBride, R. B.: The Colloidal Gold Test, J. A. M. A. **69**:1855 (Dec. 1) 1917.

Tabes Dorsalis.—In tabes, the colloidal gold reaction is generally of the 0123321100 [3] type and either this reaction or a negative result was obtained in most of the cases. However, two cases gave a paretic curve with the first lumbar puncture fluid and before any treatment was given. One other case of tabes developed a paretic curve under treatment. These cases have been under observation for two years or more and have not shown any mental symptoms.

Undifferentiated Neurosyphilis.—The cases of undifferentiated neurosyphilis are those that show no clinical signs for differentiation and yet give a positive Wassermann reaction and other findings in the cerebrospinal fluid. A group of such cases has been described by Southard and Solomon [17] under the term "paresis sine paresi." In this series of paretic curves appear the curves of seven patients with neurosyphilis who have been under observation for some time and yet have not shown any physical or mental symptoms that would warrant a diagnosis of either paresis or cerebrospinal syphilis.

17. Southard and Solomon: Latent Neurosyphilis and the Question of General Paresis Sine Paresi, Boston M. & S. J. **174**:8, 1915.

Multiple Sclerosis.—Recently, Moore [18] has shown that a paretic curve is the usual reaction of the colloidal gold test in multiple sclerosis. He found that of twenty undoubted cases of multiple sclerosis, eighteen gave typical paretic curves. Among the cases of multiple sclerosis here there were five in which paretic curves were found. One of them has been confirmed by necropsy. This patient was admitted in 1913 with most of the characteristic symptoms of multiple sclerosis. She was later sent to another hospital, and there the same diagnosis was made. The patient died in January, 1920. At necropsy numerous areas of sclerosis were found in the cord and brain. These areas were distinct even macroscopically.

The spinal fluid findings were as follows: globulin, 2; albumin, 2; cells, 7; colloidal gold, 4444332000. The Wassermann reactions on the fluid and serum were twice negative.

TABLE 5.—Cerebrospinal Fluid Findings in Five Cases of Multiple Sclerosis

Case	Globulin	Albumin	Cells	Colloidal Gold Curve	Wassermann Reaction
5025	2	2	43	5555552210	Negative
6152	3	3	0	5555330000	Negative
F. H.	2	2	2	5555431000	Negative
	2	2	4	5555421000	Negative
B. C.	2	2	8	5555531000	Negative
	3	2	8	5555421000	Negative
	2	2	14	5555521100	Negative
2223	2	2	7	4444332000	Negative

The blood Wassermann reaction was negative several times in each case.

Brain Tumors.—Brain and cord tumors generally give what is called a "chronic curve," 0011232110.[3] Many cases of brain tumor are to be found in state hospitals, but comparatively few such cases have been reported as giving paretic curves. Three cases with a diagnosis of brain tumor were found to have paretic readings in their spinal fluids, but only one of these could be definitely confirmed. This patient, whose spinal fluid gave a paretic curve on two different occasions, was found at operation to have a cerebellar tumor. The Wassermann reactions of the blood and spinal fluid were negative, and there was no evidence of syphilitic involvement of the central nervous system.

Larkin and Cornwall [12] report a case of psammona of the dura with a gold reading of 552221000. This is not a true brain tumor, and the reading is far removed from the typical paretic curve. Miller [4] found a paretic curve in a case diagnosed as cerebral gumma. It is a question whether the new growth or the syphilis or a combination of both produced this reaction.

18. Moore: Cerebrospinal Fluid in Multiple Sclerosis, Arch. Int. Med. 25:58 (Jan.) 1920.

Epidemic Encephalitis.—The reports of the colloidal gold findings in the present epidemic of encephalitis are not numerous. Almost all the cases at this hospital and most of the cases reported in the literature have given a syphilitic type of curve (1123321000). Barker [19] and his associates in their review of epidemic encephalitis report one case showing a "combined paretic and meningitic curve," and another case with an "atypical paretic curve." One case of the choreiform type of lethargic encephalitis at this hospital gave a typical paretic curve in the first fluid obtained after admission. Two tests were made with two different colloidal gold solutions and a paretic curve resulted with each.

Case 14272.—A man, aged 26, whose family history and early development apparently were normal, in the first part of 1918, had an attack of rheumatism lasting about two months. About ten days before admission to the hospital (April 21, 1920) the attack of encephalitis started with headache and pains all over the body. These pains were especially marked in the arms and back. After a few days choreic movements began, and the patient soon became delirious. Several times the patient saw double. Previous to the choreic movements the patient slept most of the time and it was difficult to arouse him. He was admitted to the hospital in a semidelirious condition. The choreic movements were marked and quite typical. The temperature was 103 and the pulse rate 104. The white blood cells numbered 16,000.

Physical examination showed marked choreiform movements of the entire body. There was a faint systolic murmur at the mitral area. The deep reflexes were exaggerated, but no pathologic reflexes were present. The pupils were equal and regular and reacted equally well to light and accommodation. Otherwise the results of the physical and neurologic examinations were negative. Mentally, the patient remained in a semidelirious condition for a few days and then gradually the mental condition cleared up. The choreiform movements were checked by chloral. For about three weeks after the chloral was stopped, the patient was restless at night but would sleep almost all day, and it was difficult to arouse him. Fibrillary twitchings of the facial muscles were often noted. The temperature remained elevated for about a month. At the end of six weeks there was marked improvement and the patient had a fair insight into his condition.

The spinal fluid findings were: globulin, 2; albumin, 2; cells, 2 (small lymphocytes); colloidal gold, 5555542100. Wassermann reactions of the spinal fluid and the blood were negative on two occasions.

This and similar cases are soon to be reported in greater detail.

Alcoholic and Drug Psychoses.—Among the cases with paretic curves in the spinal fluid appears one case of Korsakoff's psychosis (alcoholic). The diagnosis was confirmed by another hospital, and the patient was later discharged as improved.

19. Barker, Cross and Erwin: On Epidemicacite and Subacute Nonsuppurative Inflammations of the Nervous System, Prevalent in the United States in 1918 and 1919, Am. J. M. Sc. 159:157, 1920.

Case 5909.—A colored man, aged 38, whose family history and early development were normal and who had had no venereal history, had always been somewhat alcoholic, but this tendency had markedly increased during the year previous to admission. He is said to have been mentally well until about four or five days before admission when he began to complain of headache and pain in his neck. After a visit from a friend, he immediately went to the telephone to call up this friend, forgetting that he had just been there. The following day he was quite confused.

On admission to the hospital (Dec. 6, 1915) he was disoriented for time and place. He was confused, and his answers were more or less irrelevant. When asked to count backward from 20 to 1, he had to give it up after trying for fifteen minutes and getting no further than 17. Memory for recent events was quite bad. He gave the ages of his three children as 2, 3 and 8 months, respectively. No distinct fabrication was noted during his stay.

Physical examination showed a weakness of the muscles of the lower back and of the hamstring muscles, so that there was great difficulty in standing. The knee reflexes were unequal, that of the right knee being diminished. The pupils were normal, and there was no sensory disturbance.

The patient was soon transferred to another hospital from which he was discharged six months later with a final diagnosis of Korsakoff's psychosis.

The spinal fluid findings were: globulin, 1; albumin, 1; cells 2 (small lymphocytes); colloidal gold curve, 5553210000; Wassermann reactions of the blood and spinal fluid were twice negative.

In the literature many instances of colloidal gold tests in alcoholic psychoses are given, and some show mild changes in the gold, but no paretic curves are recorded.

The cerebrospinal fluid from a patient with drug (heroin) psychosis at this hospital in 1914 gave a paretic curve. This was complicated by the presence of a doubtful Wassermann reaction in both the spinal fluid and the blood. However, the patient was sent to another hospital and remained there until September, 1919, when she was discharged with a final diagnosis of drug addiction (not insane).

Lowrey [5] reports a case of drug addiction with a colloidal gold curve reading 4444100000, but there were positive Wassermann reactions in the serum and fluid. Moore [18] states that a paretic curve is sometimes present in lead poisoning. No other gold reactions resembling a paretic curve could be found in many of the cases of drug addiction reported, although many did show mild changes in the syphilitic zone.

Undiagnosed Cases Definitely Not Paresis.—There were five other cases in which a definite diagnosis could not be made, but in which paresis could be ruled out.

Case 4398.—The mother and several brothers of a white man, aged 40, died of tuberculosis. One sister was insane. The onset and course of the patient's illness was very indefinite. On admission (Feb. 21, 1915) he was found to be suffering from advanced pulmonary tuberculosis. There was involvement of the entire left lung and the upper and middle lobes of the

right lung, with cavity formation. The neurologic examination was negative. The patient slept most of the time. Temperature, pulse and respirations were increased. Memory was poor for recent and remote events. His grasp on his surroundings was poor. No hallucinations or delusions were found. The patient was unemotional and had no insight. He gradually grew worse and died eleven days after admission. No signs of meningeal involvement were found at any time and a necropsy examination was not obtained; therefore the diagnosis of tuberculous meningitis, which is the most probable one, could not be confirmed. A guinea-pig was inoculated, but owing to contamination no results were obtained.

The spinal fluid findings were: globulin, 3; albumin, 4; cells, 69 (small lymphocytes. 68 per cent., large lymphocytes, 22 per cent., plasma, 3 per cent., polymorphonuclears, 7 per cent.). The colloidal gold curve was 5555554321. The Wassermann reactions in the fluid and serum were twice negative.

Cases of tuberculous meningitis with paretic curves have been reported as shown in Table 6.

CASE 13364.—A white woman, aged 49, whose family history was negative, was healthy until 12 years old when she developed curvature of the spine; since then she has worn braces occasionally. The patient graduated from normal school when she was 21 and was considered a good student. She was somewhat of a "book worm." When about 28 years old she began to prepare to get married. She had a wedding dress made and even resigned her position, but there was no man in the case. The patient was at that time sent to a state hospital and remained there two months. She has not been normal since, according to her friends. She has a circle of friends whom she calls her "arch friends" and who have a "program" of persecution. After making several attempts to kill one of them she was sent to this hospital for observation (Oct. 14, 1919).

On admission her mental condition showed nothing abnormal except a wealth of paranoid ideas that she had maintained for twenty years. Among her "arch friends" was a man or male spirit whom she prepared to marry twenty years ago. Since then this spirit has had sexual intercourse with her by means of electricity. The patient had a religion all her own in which her hands play an important part. Auditory and visual hallucinations were denied. She had some insight at times. Personality was well retained.

Physical examination revealed a right upper thoracic scoliosis and a resulting deformity of the chest. There was a scar on her right breast where the entire right mammary gland was removed a few weeks before entering this hospital. There was a lump the size of a walnut in the left breast and palpable axillary glands. The neurologic examination was negative.

After four months the patient was transferred to another hospital from which she was later discharged as improved. The diagnosis remained "paranoid condition."

The spinal fluid findings were: globulin, 1; albumin, negative (?); cells, 3 small lymphocytes, 1 large lymphocyte; colloidal gold curve, 5555531000 (two tests). The Wassermann reactions on the spinal fluid and serum were twice negative.

CASE 13179.—A white man, aged 26 (?), whose family history was somewhat defective, was a full term, healthy baby but could not talk until he was 4 years old. He had smoked excessively since he was 8 years old. He was quarrelsome and mischievous as a child, but quite honest. He was backward

in school and did not learn to read very well. He repeated the fourth and fifth grades and never got much further. At 13 he ran away from school. In 1914 he was arrested for stealing and sent to this hospital. Because of an apparent character change and abnormal emotional reaction the condition was diagnosed as dementia praecox simplex, and the patient was transferred to another hospital. After three months there he was discharged and went back to work as a machinist. His mental age at that time was 11.1. In 1917 he enlisted and was sent to France. He was returned to this country in January, 1919, after being gassed twice, and was placed at various base hospitals. Finally, he remained away without official leave for eleven days and was sentenced to serve three years. After serving four months he was again sent to this hospital for observation.

On admission (Sept. 8, 1919) he was correctly oriented. Memory was somewhat bad and the details of his story did not check up well. Calculation ability and school knowledge were rather poor. No evidence of delusions was found, but he admitted hearing bells and voices while in France. Emotional responses were quite normal, and personality was well retained. There was no disturbance of thought processes. His mental age was not tested again. Physically, he was well developed and well nourished. His breath was foul, and the tongue was tremulous, rough and fissured. His hands and feet were cold and cyanotic. The pupils were irregular and reacted sluggishly to light and accommodation through a narrow arc. The knee reflexes were sluggish but equal. Sensation was normal. There was no Romberg sign.

The spinal fluid findings were: globulin, 1; albumin, 1; cells, 41 small lymphocytes; colloidal gold curve, 5554432100. The colloidal gold test was repeated with a different colloidal gold solution, and practically the same result was obtained. The Wassermann reactions on the serum and spinal fluid were consistently negative several times. The patient was given provocative arsphenamin, but the Wassermann test remained negative. After the provocative treatment another lumbar puncture was made with the following results: globulin, 1; albumin, 1; cells, 20 small lymphocytes; colloidal gold curve, 0123220000.

Since being discharged the patient has gone back into the transport service and is getting along well. The blood Wassermann test remains negative.

Case 13430.—The patient gave a history of excessive alcoholism for several years before admission to the hospital with an increase in the amount of alcohol consumed just prior to entrance. A history of a chancre about a year and a half before admission complicated the case. The patient became hallucinated about six months before he was sent to the hospital. On entrance he had marked hallucinations and delusions. The hallucinations were auditory and were mostly in the third person. He thought that he was persecuted by the Catholics and that they had electromagnets which controlled his thoughts. There were numerous other delusions of persecution. There was no obvious disintegration of personality and no memory loss. The patient was superior in the psychometric test. Orientation was intact, and there was no disorder of consciousness. Emotional tone was in keeping with his delusions. The physical and neurologic examinations were entirely negative. After six weeks in the hospital the hallucinations had almost completely disappeared, and the patient had good insight.

The spinal fluid findings in the first fluid tested were: globulin, 1; albumin, 1; cells, 8 small lymphocytes; colloidal gold curve, 5555321000. The Wassermann test was negative. The patient was given several intravenous injections

of arsphenamin, but the blood and spinal fluid Wassermann reactions remained negative, while the other spinal fluid tests were practically the same on two subsequent examinations.

Case 5364.—A girl, aged 23, admitted to this hospital in 1915, was of the defective delinquent type, and this was the provisional diagnosis, although her case was later left unclassified. She was moody, depressed and easily irritated. She had delusions of persecution, but no hallucinations. Physical examination revealed only poorly reacting pupils. The diagnosis of manic depressive insanity was considered, and it is possible that it was a case of neurosyphilis although the Wassermann reaction was negative. The patient was sent to another hospital from which she was later discharged as improved.

The spinal fluid findings were: globulin, 2; albumin, 2; cells, 13; colloidal gold curve, 5555543310. The Wassermann test was negative.

TABLE 6.—Miscellaneous Cases; Neurosyphilis and Multiple Sclerosis Excluded

Author	Number of Cases	Source or Type of Cases	Paretic Curves	Conditions
Warwick and Nixon [20]	245	General hospital	1	Brain abscess
Lowrey [5]	115	State hospital	1 (?)	Drug addict
Larkin and Cornwall [21]	114	Epilepsy	1	Epilepsy
Stevens [22]	38	Mongolian idiocy	0	
Hammes [8]	92	General hospital	0	
Jeans and Johnson [23]	100	Poliomyelitis	0	
Weston [14]	165	State hospital	2	Undiagnosed
Vogel [24]	52	General hospital	1	Questionable
Rawlings [7]	319	Psychopathic hospital	1	Defective
Williams and Burdick [25]	79	.....	0	
Swalm and Mann [6]	27	.....	0	
Miller, Brush, Hammes and Felton [4]	102	Psychopathic hospital	2	Brain abscess; tuberculous meningitis
Eskuchen [11]	56	.....	0	
Jaeger and Goldstein [10]	54	.....	3	Brain abscess; tuberculous meningitis; epilepsy
Eicke [9]	222	.....	0	
Larkin and Cornwall [12]	115	.....	4	Eclampsia, 2; psammoma of
Present series	6,500	Psychopathic hospital	9	Cerebellar tumor, 1; Korsakoff's psychosis, 1; encephalitis, 1; drug addict, 1; tuberculous meningitis, (?) 1; undiagnosed, 4

20. Warwick and Nixon (Footnote 2).

21. Larkin and Cornwall: Spinal Fluid in Epilepsy, J. Lab. & Clin. Med. **4**:352, 1919.

22. Stevens, H. C.: The Spinal Fluid in Mongolian Idiocy, J. A. M. A. **66**:1373 (April 29) 1916.

23. Jeans and Johnston: Am. J. Dis. Child. **13**:329, 1917.

24. Vogel, K. M.: The Nature and Interpretation of the Colloidal Gold Reaction, Arch. Int. Med. **22**:496 (Oct.) 1918.

25. Williams and Burdick: Lange Colloidal Gold Reaction, Colorado Medicine **13**:103, 1915.

Rawlings [7] reports a case with a paretic curve in which the diagnosis was "defective."

In the literature from other similar hospitals, from general hospitals and from army hospitals, other conditions giving rise to paretic curves were found. These, together with the cases from this hospital, are given in Table 6, which gives the miscellaneous cases reported,

excluding neurosyphilis and multiple sclerosis, because paretic curves are to be expected in such cases.

VARIOUS OTHER CONSIDERATIONS

Relation to Other Tests.—The nonconcomitance of spinal fluid tests has been fully discussed by Solomon.[26] He concludes that any one of the usual five tests may be present or absent when the others are present, because each reaction is produced by a distinct chemical element that may be present alone. The relation to the Wassermann test is shown in Table 3 and has been discussed by Hammes.[8]

Effects of Treatment.—The colloidal gold reaction may vary considerably under treatment just as the other spinal fluid and blood tests do. With intensive treatment in favorable cases the tendency is for the paretic curve to become irregular or change to the "syphilitic type" and then finally to become negative. We have many such instances of which the following is a good example:

TABLE 7.—VARIATION OF COLLOIDAL GOLD REACTION UNDER TREATMENT*

Date	Colloidal Gold Curve	Globulin	Albumin	Cells	Wassermann Reaction	
					Cerebrospinal Fluid	Serum
3/21/15	5555554321	4	4	80	Positive	Positive
3/31/15	5555553100	4	4	96	Positive	Doubtful
4/ 7/15	5555543200	3	3	67	Positive	Doubtful
5/ 8/15	0011100000	2	1	8	Doubtful	Doubtful
8/13/15	1000000000	1	1	5	Negative	Negative
10/21/15	0000000000	0	Normal	4	Negative	Negative

* Intensive intraspinal and intravenous treatment.

The time of disappearance of the paretic curve is variable as has been pointed out by Solomon.[26] According to Neyman and Frush,[27] the paretic curve is the last of the positive spinal fluid reactions, with the exception of the globulin test, to disappear under treatment.

Improvement in the paretic curve is not the rule in treated cases. In fact, the reaction may become more pronounced either temporarily or permanently, just as the Wassermann reaction has been observed to do. The changes in the colloidal gold test may not parallel the clinical change in a treated patient. At present the paretic curve, as well as the other reactions of a patient who is having an almost perfect clinical remission, remain strongly positive in spite of intensive treatment. The opposite condition has also been found in a few instances. However, when there is clinical improvement and the other tests tend

26. Solomon, H. C.: Nonconcomitance of Spinal Fluid Tests, Arch. Neurol. & Psychiat. **3**:49 (Jan.) 1920.

27. Neymann and Brush: Treatment of General Paralysis, Arch. Int. Med. **22**:245, 1918.

to clear up, the gold reaction will, in the great majority of cases, follow the same course.

Provocative Colloidal Gold Test.—McBride [16] made the statement that there is no colloidal gold reaction to provocative treatment but, on the other hand, Warwick and Nixon [20] and others speak of finding a provocative colloidal gold reaction. We have several cases in which the colloidal gold reaction was at first negative, but after a few treatments, a typical paretic curve appeared and remained for some time. This has happened too often to be merely coincidental. The cases in Table 8 are illustrative of this, and it will be noted that the other findings may also become more marked.

TABLE 8.—COLLOIDAL GOLD REACTIONS TO PROVOCATIVE TREATMENT

Date	Colloidal Gold Curve	Globulin	Albumin	Cells	Wassermann Reactions		Treatment
					Cerebrospinal Fluid	Serum	
Case 13013	.....	...	...	....	.....	.....	Between dates given, 33 intravenous injections and 7 grains of mercury; no previous treatment
1/24/19	0011230000	?	?	139	Negative	Positive	
10/ 4/19	5444310000	1	1	30	55.0000	Negative	
Case C. A.	.....	...	...	....	.....	.....	No previous treatment: between dates given, 19 intravenous injections and 11 grains of mercury
1/15/19	0000000000	0	N	2	Positive	Positive	
1/20/20	5555531000	1	2	2	5555000	Positive	
Case W. F.							
11/18/15	1233330000	3	3	20	55520	Negative	3.3 gm. arsphenamin
12/15/15	5555553100	3	2	12	50420	Doubtful	2.2 gm. arsphenamin
2/24/16	3333220000	2	2	2	Doubtful	Negative	1.8 gm. arsphenamin
1/26/17	0000000000	1	1	2	53111	Negative	
Case W.Mc.	.....	...	...	....	.....	.....	No previous treatment
6/14/19	0000001010	0	N	0	Negative	Negative	1.6 gm. arsphenamin
8/ 2/19	5555431000	1	1	13	Negative	Negative	1.2 gm. arsphenamin
9/ 6/19	5444321000	1	1	7	Negative	Negative	
10/11/19	5433211000	1	1	1	Negative	Negative	
Case E. F.							
8/10/18	1112321000 1114321000	2	2	116	500000	Positive	0.8 gm. arsphenamin and 1 intraspinal injection; 4 gm. arsphenamin
12/ 2/18	5554443221	1	1	2	555540	Positive	
11/ 2/19	5554443210	1	1	3	555550	Negative	
Case F. M.							
5/15/18	0001332110	2	1	56	.....	Doubaful	8.4 gm. arsphenamin
3/15/19	1222333310	0	N	2	5000000	Negative	2.3 gm. arsphenamin
8/ 9/19	5433310000	0	1	7	2200000	Negative	2.6 gm. arsphenamin
1/24/20	0111110000	1	1	5	Negative	Negative	

It sometimes happens that without the usual treatment a paretic curve may appear after three or four lumbar punctures. This may in some cases be coincidental with the development of the disease, or it may be that the repeated lumbar punctures act as a provocation.

Effect of Contamination.—Various contaminations of the cerebrospinal fluid may modify the paretic curve. The fluid may become contaminated by using dirty test tubes or old corks that have been used with drugs; by the growth of yeasts or molds; by blood obtained when the lumbar puncture is made; and by various other foreign substances. In general, such contaminations may change a syphilitic type of fluid into a paretic type, but will seldom if ever produce a paretic curve in an absolutely normal fluid. A paretic fluid that has become contaminated will usually give a somewhat irregular reading resembling a paretic curve and may become practically negative.

With due care all these sources of contamination may be avoided with the exception of blood, which will sometimes be obtained in spite of all precautions. After repeated experiments of adding various quantities of blood to known normal fluids, it was found impossible to produce a reading that could be called a paretic curve. Such fluids would show marked and irregular color changes in the middle or at the end of higher dilutions. Addition of blood to the syphilitic type of fluid would occasionally give a paretic curve. Blood added to a known paretic fluid would generally produce an irregular reading but not a negative one. However, in cases contaminated at the time of puncture, paretic curves have been noted in normal cases and vice versa.

The effects of the presence of blood in the cerebrospinal fluid has been discussed by Black [16] and Kellert.[28]

CONCLUSIONS

1. The paretic gold curve does not always indicate paresis, but, if used in conjunction with the other spinal fluid tests, it is a most valuable test for this disease. In some instances, this reaction is the deciding factor in the diagnosis of general paralysis.

2. The vast majority of cases of paresis will give a paretic type of gold reaction. In a case that does not give such a reaction after more than one examination, the diagnosis of paresis is always questionable.

3. The presence of paretic curves in almost 50 per cent. of the cases of cerebrospinal syphilis shows that a paretic curve does not serve to differentiate paresis from this condition. However, the absence of a typical reading points strongly to cerebrospinal syphilis when the differential diagnosis lies between these two conditions.

4. A paretic or marked syphilitic curve is to be expected in multiple sclerosis.

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28. Kellert: Observations on Colloidal Gold Reactions with Cerebrospinal Fluid, Am. J. M. Sc. **159**:257, 1920.

5. Besides paresis and multiple sclerosis, a paretic curve is occasionally found in the following conditions: tabes dorsalis, cerebrospinal syphilis, undifferentiated neurosyphilis, brain tumor, brain abscess, encephalitis, tuberculous meningitis, Korsakoff's psychosis (alcoholic), eclampsia, epilepsy, lead poisoning (?) and drug addiction (?).

6. The pathology of the above conditions would indicate more or less destruction of nerve cells in the brain, or, in other words, a parenchymatous involvement. It may be concluded, therefore, that a paretic curve points toward parenchymatous involvement of the brain, while the milder gold curves are obtained in meningitis, vascular disease and other conditions. The paretic curve is not only of diagnostic value, but also of prognostic value.

DEGENERATION GRANULES IN BRAIN CELLS IN EPIDEMIC ENCEPHALITIS*

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In the routine examination of sections from the brain of a case of epidemic encephalitis, minute granules were found in degenerated nerve cells in the areas involved by the disease process. Although similar granules in many diseases of the central nervous system have been described in German publications, notably by Alzheimer [1] and Achucarro,[2] to my knowledge their presence in cases of epidemic encephalitis has not previously been noted, and it therefore seems worth while to describe them as part of the microscopic pathology of this disease.

The granules are best demonstrated in tissues fixed in Zenker's or Helly's fluid, embedded in paraffin and stained with eosin-methylene blue or by Bensley's modification of the Altmann stain (acid-fuchsin-methyl-green). To bring them out with eosin we used a combination of equal parts of strong alcoholic and 5 per cent. aqueous eosin, staining forty minutes at 56 C. This stain, made up by mistake for routine eosin-methylene blue staining, was the one with which we first demonstrated the granules. With it the red blood cells in the sections appeared a deep copper red, the stroma bright pink, and the granules bright red or purple red. With Bensley's stain they appeared a somewhat brighter red.

As demonstrated by these stains, the granules appear as minute spherical masses, measuring from about 0.5 to 2 microns in diameter, sharply outlined and highly refractile. The largest ones are nearly half as large as the nucleoli found in these nerve cells, but they are not so large as even the smallest Negri bodies of rabies, which stain somewhat similarly with acid fuchsin. The larger ones often have a minute colorless center, as though the stain had not thoroughly penetrated. They also are frequently surrounded by a clear zone in the cell cytoplasm. In number they vary greatly, and their number

* From the Medical Pathological Laboratory of the Presbyterian Hospital, Department of Pathology, College of Physicians and Surgeons, Columbia University.

1. Alzheimer, A.: Histologische und Histopathologische Arbeiten **3**:491, 1910.

2. Achucarro, N.: Histologische und Histopathologische Arbeiten **3**:174, 1910.

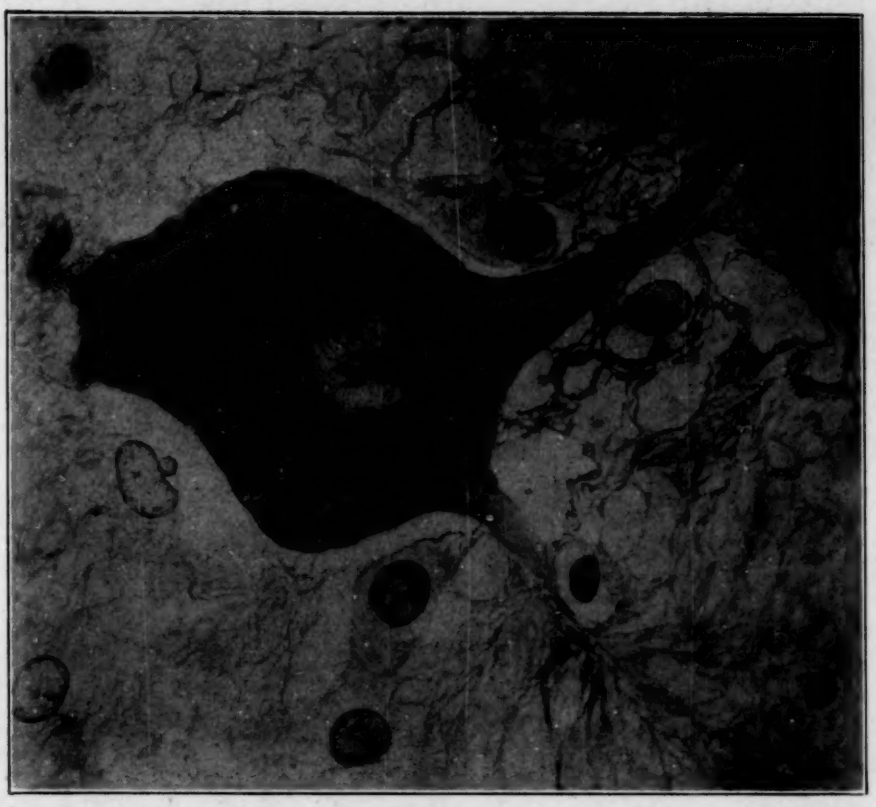

Fig. 1.—Degenerated ganglion cell from a basal ganglion of a case of epidemic encephalitis, showing degeneration granules in the cytoplasm.

Fig. 2.—Smaller ganglion cells from the same section, showing degeneration granules and beginning neuronophagia.

appears to have no relation to the degree of degeneration of the nerve cell in which they occur. Frequently only a single granule is found in a cell, while other cells, whose appearance is no more abnormal, contain forty or fifty granules. Some definitely degenerated cells contain no granules in the section studied, though parts of the cell not cut may have contained them. The nerve cells in which these granules are found are always more or less abnormal in appearance, as evidenced by loss of definite outline, dissolution of Nissl substance, acidophilic reaction of cytoplasm—particularly in the region of the granules—accumulation of lipochrome pigment in the cytoplasm, swelling or fragmentation of the nucleus, or beginning phagocytosis of the nerve cell by invading wandering cells. The degenerated cells are found only in areas in which there are other lesions characteristic of the disease, namely, perivascular hemorrhage, or perivascular or diffuse lymphocytic infiltration. In the cases of epidemic encephalitis studied, granules have been found in the occipital cortex, the basal ganglions,

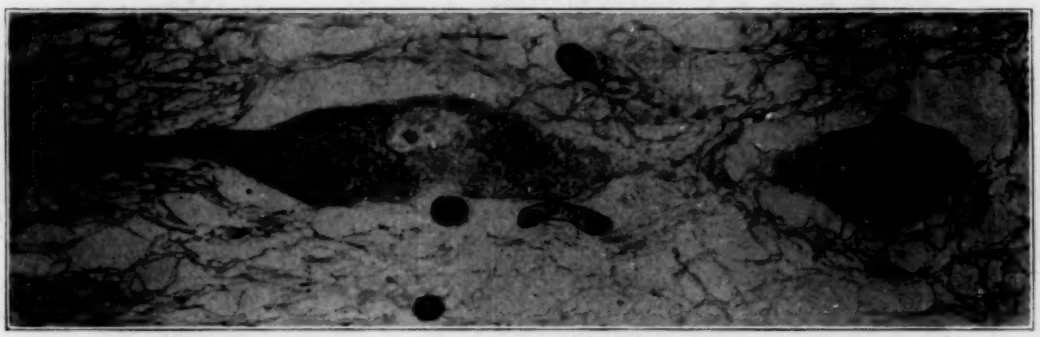

Fig. 3.—Ganglion cells from the basal ganglions of another case of epidemic encephalitis, showing degeneration granules, some of which are outside the cell due to partial lysis of the cytoplasm.

the pons and the medulla. They have not been found in the anterior horn cells of the spinal cord, although one case showed marked perivascular lymphocyte infiltration in the spinal cord, and definite degeneration of anterior horn cells. In the nerve cells of the posterior root ganglions of the same case granules were found which gave the same staining reaction as the granules I am describing, but they were more irregular in outline, were more numerous and were usually arranged in small rosettes.

No extensive study has been made of the chemical nature of the granules. Sections treated with 10 or 20 per cent. nitric acid for five minutes before staining show the granules as brilliantly as before, whereas sections similarly treated with 5 per cent. sodium hydroxid show them hazily outlined and dull red in color. Sections fixed in

liquor formaldehydi show the granules poorly or not at all, the acid-fuchsin stain bringing them out better than eosin.

That the granules are not specific to epidemic encephalitis is shown by the fact that they have been demonstrated in the basal ganglions, pons and medulla, and once in the spinal cord in four cases of poliomyelitis, and in the basal ganglions in a case of tuberculous meningitis associated with lymphocytic infiltration of the brain stem. Granules similar to those found in the dorsal root ganglions of the case of epidemic encephalitis mentioned were also found in the dorsal root ganglion cells of a case of tabes dorsalis. On the other hand, several cases coming to necropsy, which showed no clinical or anatomic involvement of the central nervous system, have failed to show these granules in the nerve cells of the brain.

Without more extended study it is perhaps unwise to discuss the possible derivation of these bodies. Their constant association with degenerative changes in the nerve cells indicates that they are some form of cytoplasmic degeneration. The most obvious source, because they stain with mitochondrial stains, would be the mitochondria themselves. The studies of McCann [3] on the posterior root ganglions of poliomyelitic monkeys have shown that the mitochondrial filaments may persist in the presence of pathologic changes in the ganglion cells. The granules described in this paper, if derived from mitochondria, are necessarily much altered in their chemical structure, as they are resistant to postmortem autolysis and to the action of strong acid and alkali, and are demonstrable by staining methods which do not bring out mitochondria in the same cells.

3. McCann, G. F.: J. Exper. Med. **27**:31, 1918.

THE PSYCHIATRIC FEATURES OF SO-CALLED LETHARGIC ENCEPHALITIS

CASE REPORTS AND A REVIEW OF THE LITERATURE

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Probably no single topic in the field of nervous and mental diseases has, within the past year, received more attention than so-called lethargic encephalitis, and yet the vast literature on the subject contains little concerning the psychiatric aspect of this disorder besides that given in a few papers noted later under a discsussion of the literature.

We came in direct or indirect contact with about fifty or more cases of this disorder during the winter and spring of 1920. With the exception of the four cases reported in this paper, especial attention was not given to the mental picture except in regard to the condition called "substupor" that was so pronounced in most of the cases. The physicians who had charge of most of the patients were more particularly interested in cranial nerve palsies, lost knee jerks, spasmodic muscular twitchings, bladder disturbances, nystagmus and a host of other neuropathologic findings. We present this subject because we studied the mental conditions of our patients and because three, if not all, of our cases occurred in persons who had, before their infection, exhibited more or less pronounced neurotic tendencies. One had been under observation for some time to determine whether he was suffering from so-called petit mal epilepsy or from anxiety attacks of hysteria. Another had an antecedent history of obsessions; a third was a highly unstable and emotional child, and the fourth was of distinctly high strung temperament. This raises the question, perhaps of a will-o'-the-wisp character, as to whether the neurotic tendencies predisposed the patient to an attack of encephalitis or whether the attack threw into bold relief the psychiatric picture in the case.

REPORT OF CASES

CASE 1.—*History.*—B. B., a white boy, aged 9; whose maternal grandmother died at the age of 53 of a "nervous skin eruption" and whose paternal grandfather died of apoplexy at the age of 69, had been a patient at Pontiac State Hospital for some time. His mother, aged 29, was emotionally unstable. The father was apparently normal, a successful salesman. He was suing the

patient's mother for divorce on the ground that her youngest child (10 weeks old at the time the patient was first seen) was not his. The mother was bringing counter charges of infidelity.

Gestation and birth were negative. He had had measles, mumps and whooping cough after beginning school. During the past three or four years he had always become fatigued easily on exertion, and on several occasions had had episodes of sleeping for two or three days following exertion.

Make-Up of the Patient.—He was easily controlled and of a happy disposition, with the exception of frequent spells of emotional instability not unlike those of the mother. He was fond of boyish sports. His mentality was above the average; this was confirmed by intellectual tests.

Present Illness.—He contracted what was diagnosed by the family physician as influenza in February, 1920. His temperature was elevated for eleven days. Recovery was good except for "lameness" in the right leg and knee. This condition was intermittent. He had been out of school twenty-three half days. He returned to school about March 8, doing well until April 14, 1920, when his teacher noticed that he was slow in arithmetic, and that his handwriting was tremulous to the point of being illegible. At this time the mother noticed that he continually tossed about in bed with movements not unlike those of acute chorea. Intention tremor was noticed especially when he tried to eat or drink. He had to be fed for this reason. He was unable to button his clothes. His speech was thick. He had progressive emotional instability.

Examination.—The patient was first seen April 19, 1920. His face was flushed. The temperature was 100.5 F.; pulse rate, 108. Blood pressure was systolic 95, diastolic 58. The heart beat was irregular and pounding, but no murmurs were detected. Neurologic examination showed wide choreiform movements of all four extremities. The throat was difficult to examine because the head was in constant motion. Knee reflexes were not obtained. The chief mental symptom was emotional instability.

Clinical Course.—April 20, lumbar puncture showed a clear fluid, twenty-one cells, negative globulin and a flat gold curve. There was moderate leukocytosis and 13,500 white cells were found. Blood culture was negative. There was a cell count of twenty-three, and of nineteen on the occasion of two later spinal punctures made within the following week. After that the spinal fluid was negative on two occasions and within two weeks the blood count was normal, and all neurologic symptoms had disappeared, except choreiform movements of the hands, and the absence of knee reflexes. These in turn disappeared within a month after the patient was first seen.

Mental Picture.—The outstanding feature, both before and after the patient came under the writers' observation, was marked emotional instability. There never was a period or stage that could be designated as the lethargic or substuporous stage. On the third day after he was examined there was a short period of unclearness or mental confusion. After one of his evening meals he appeared half delirious, getting out of bed, and then trying to get in again. He appeared to realize that he had defecated in the bed, but seemed to have lost the power of speech, only pointing to the soiled linen. When power of speech returned there seemed to be a transient aphasia, or paraphasia. For this episode he later had no memory. He was discharged as recovered, six weeks after he was first seen. At present he shows no evidence of his illness except a slight emotional instability.

Had it not been for the spinal fluid findings, the leukocytosis, and the temporary loss of the patellar reflexes, the case would have been regarded as one of Sydenham's chorea.

The outstanding mental symptoms were: emotional instability, inability to perform simple arithmetical calculations, and a brief period of unclearness with amnesia for the same on recovery. There was no evidence of mental deterioration or defect as a result of the illness. It should not be forgotten that the patient was emotionally unstable during a state of physical health, and that there was a taint in the family history.

CASE 2.—*History.*—A. M., a young man aged 28, single, white, a machinist, had a negative family history. The previous medical history was unimportant.

Make-Up of the Patient.—He was somewhat high strung. He always worked as though under tension, frequently late into the night with his books, even after long hours in the factory as an automobile mechanic. He frequently talked of inventions and machinery. He was fairly sociable in his habits; somewhat sentimental.

Present Illness.—On Tuesday, April 13, 1920, the patient began to be troubled with terrifying dreams, such as concerning a bear getting under his bed, and creeping up the bed after him. He complained of general malaise, but felt the necessity of keeping at his work. He obtained little or no sleep for the next four nights. He had anorexia, and was troubled with nausea and vomiting on the way to, or at, work.

Absentmindedness was noticed by his landlady. He would start to work without his door-key, papers, pencils, etc. On Tuesday, April 15, he started to work in the rain without his umbrella. He returned to the back stairway for his rubbers when they were in the front hallway. Choreiform movements began that night. He had subjective sensation of teeth falling out, relieved only by biting the hot-water bottle.

For the week preceding these symptoms the patient had been troubled with sore throat. Friday, April 16, frequent yawning and stretching movements, difficulty in breathing and ambling gait were noticed. That night generalized threshing or tossing movements of entire body were present. The family physician was called. The movements were not affected by morphin, ¾ grain, or veronal, 30 grains, in twenty-four hours.

Examination.—The patient was first seen on Sunday, April 18, at noon. His face was flushed, and he was tossing violently about in bed, his movements being not unlike those of one having a severe hysterical attack. He would throw himself in the air, the rebound almost severing the springs from the bed. He answered questions, however, and said he suffered no pain. He kept his eyes closed most of the time. He could be quieted by laying a hand on his trunk or extremities.

Neurologic examination disclosed nothing unusual. Pupillary responses, and extra-ocular movements were normal. There was no nystagmus. Knee reflexes were normal, as were the other deep tendon reflexes. The superficial reflexes were unimpaired. Blood pressure was normal. The temperature was 99 F. He was admitted to Harper Hospital on April 18, 1920. For the next two days threshing movements continued, at times almost becoming opisthotonos. At all times he responded rationally to questions.

On the third day the knee reflexes were questionably absent and the abdominal reflexes were not obtained. There was a bilateral weakness of the sixth nerve, more pronounced on the left. Morphin had no effect on the spasmodic

movements. Apomorphin did not cause vomiting, but did have a quieting effect. The laboratory findings were:

1. Spinal fluid: negative Wassermann reaction; cell count, 140; decided increase in pressure; crystal clear fluid; globulin, positive; mastoid and gold curves negative.

2. Blood: negative Wassermann reaction; blood nitrogen, 29 mg. per 100 c.c.; blood count: red, cells, 4,650,000; white cells, 9,000; differential count, negative; red cells, normal in size and staining reaction; hemoglobin, 80 per cent.

3. Urine: negative.

On the first day of admission to the hospital, the temperature rose to 102 F., and gradually fell to normal by the fourth day.

Mental Picture.—He had terrifying dreams and was unable to concentrate. He responded well to questions. On the day of his admission to the hospital, insight was poor, the patient regarding himself as well. As proof he stated that he was no longer troubled with certain numbers running constantly through his mind, such as "16 Z 1185," a number on a machine part which he had been making, or "167753," the engine number of his automobile. "If I had nothing to read, I would keep repeating these numbers. I was sick then; I am well now." On April 20, the patient stated that he had a total amnesia for a period of four days from April 16 to 19 inclusive, and he did not remember his terrifying dreams, the visits of his physicians, or his admission to the hospital. During the period of his amnesia he frequently manipulated his genitals, even when the nurse was in the room. Of this he also had no memory, but he thought he had had a diplopia for a few minutes on the night of the 16th when he was first taken acutely ill.

Clinical Course.—After the fourth day in the hospital, improvement, both mentally and physically, was rapid. For a week there was a period of mild drowsiness from which he could be easily aroused. He was discharged one month after admission. At that time the chief residual of his illness was a slight blurring of vision. He could not read for over twenty minutes without fatigue. There was still a noticeable weakness of the external recti and a slight arrhythmia of the heart. Three months after his discharge from the hospital, he was able to return to work, and the only residual of his previous illness was a drowsiness which usually appeared about 9 o'clock in the morning while he was at work. "Even a few seconds of dozing" would make him "good for the rest of the day."

The principal mental symptoms in this case are:

1. Terrifying dreams.

2. Absent-mindedness and diminished power of concentration.

3. Delirium of a mild character with complete amnesia for the period of delirium. This period was marked by spasmodic twitchings and generalized movements, manipulation of the genital organs, mild degree of euphoria and lack of insight.

4. Short periods of substupor immediately following the irritative stage. This substuporous condition was not marked. He answered questions readily, but usually dropped off into a half drowsy state after the questions were answered. This period continued for about a week. At no time was it difficult to get his attention.

5. Three months after his discharge from the hospital there were brief moments of this drowsy tendency, occurring usually in the morning.

Case 3.—*History.*[1]—S. O., a single man aged 23, a Russian Jew, had a negative family history, except for the fact that he and many of his brothers and sisters, nine in number, suffered from enuresis, several as late as the sixteenth year. He came to this country from Russia at the age of 15. He had received a high school education. For several years he had assisted an older brother in the mercantile business, and for three years he had managed a small store himself.

About 1915, the patient began to have attacks, or "spells," of an epileptiform or hysteroid character. They would usually come on in the morning just after he had arisen, and would last not longer than a minute. There were aura, particularly peculiar sensations and paresthesias in the right arm. He never fell, but was able to get to a chair or bed. He would then become tense and rigid and appeared to lose consciousness for a short interval. He was admitted to the State Psychopathic Hospital at Ann Arbor, for observation, on Nov. 19, 1917, and was discharged December 11 of the same year.

Physical examination was practically negative. Neurologic examination showed a myopic fundus with a congenital choroidal lesion in the left eye. There was questionable horizontal nystagmus. There was a slight facial asymmetry. During the examination, muscles of the thighs and calves showed twitching and both limbs exhibited tremors. Patellar and Achilles' reflexes were active.

Laboratory findings, including the blood Wassermann reaction, were negative.

During his residence at Ann Arbor the patient had about four mild convulsive seizures. All occurred during the night and left no bad after-effects. On Dec. 8, 1917, he had several seizures of mild character with momentary confusion, accompanied by slight general muscular twitchings. His case was diagnosed as epilepsy, nocturnal petit mal attacks.

Make-Up of the Patient.—He was quick-tempered, but sociable and popular with his associates. For two years his habits were somewhat irregular and a source of concern to his family. He denied venereal infection.

Present Illness.—On Dec. 31, 1919, the writer was called in consultation. It was learned that his so-called petit mal attacks had gradually lessened in frequency after his discharge from Ann Arbor, and that for three months prior to December 31, these attacks had disappeared entirely. Even before that time an attack could be aborted by awakening him during the night or by simply turning on the electric lights in his room.

On or about Dec. 17, 1919, the patient had caught cold following the opening of some new stores. He appeared to recover, but had a slight relapse, and was confined to bed a day or two about December 22. On Christmas day he began to suffer what was described as "neuritis" affecting one part of the body, then another, but never the joints. He was sent to Mount Clemens for the baths, but while there he grew steadily worse, the chief symptom being severe spasmodic twitchings of the muscles of the trunk, which the brother thought might be "hysterical."

On Jan. 21, 1920, he was admitted to Harper Hospital in the Neurological Service.

On the day of admission the neurologic examination was negative, except for irregular jerking movements of the abdominal muscles.

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1. History of this case from Nov. 19, 1917, to Dec. 11, 1917, was obtained by courtesy of Dr. Albert M. Barrett, Director of State Psychopathic Hospital, Ann Arbor, Mich. S. P. H. No. 2191.

Mentally he appeared to be in a dream state or a mild delirium. He constantly kept his eyes closed and emitted grunting noises not unlike those of an animal. He was restless, and continually picked at the bed clothes; at other times he would cover his head with the bedspread not unlike a manic or praecox patient. He would frequently ask for the urinal, get out of bed in a half-dazed way, stand as if puzzled, and would not urinate. When asked where he was suffering pain, he would answer, "Six dollars." Three tenths of a grain of apomorphin was given with no apparent effect.

On January 2, he was disoriented for time, person and place. One physician was a "policeman," and the other a "mail collector."

The neurologic examination was still negative on January 2, but lumbar puncture gave a positive globulin test and 90 cells in the fluid. Other important laboratory findings, January 7, were:

1. Urine, negative.
2. Blood count: red cells, 4,900,000; white cells, 14,800.
3. Blood Wassermann, blood culture and blood nitrogen, all negative.
4. Spinal fluid Wassermann and culture negative.
5. Lange and Mastic curves negative.

From that time on the patient's symptoms became more grave. His temperature began to rise, reaching as high as 104 F. The spasmodic contractions of the abdominal muscles continued; the right pupil became sluggish; the tendon reflexes, especially the patellars were somewhat delayed; there was a noticeable intention tremor, and speech became somewhat thick. Cardiovascular and respiratory symptoms appeared, and it became necessary by January 10 to inject normal salt solution intravenously. For several days before this the "substuporous" mental state appeared, or perhaps what might better still be called a "low grade stupor," from which the patient could always be aroused sufficiently to answer questions, though his mentality was as a rule unclear. This stupor was more marked in the morning.

On January 5, the patient's spinal fluid agglutinated the blood of five or six other similar cases then in the hospital, and this was regarded as a favorable symptom. Nevertheless he steadily grew worse. His condition became so bad that two blood transfusions were done, the donors being his brothers. In spite of this he died in a state of coma on January 26, twenty-six days after admission. Unfortunately, necropsy was refused by the family.

It should be added that during the last two weeks of his illness bladder symptoms appeared, the urine being literally ejaculated frequently the full length of the bed.

Mental Picture.—The principal mental features in this case are:

1. Several siblings with enuresis up to as late as the sixteenth year.

2. The patient was subject to petit mal attacks of nocturnal epilepsy for three or four years before his encephalitic condition. These attacks could be aborted by simple measures.

3. Early in his acute illness he appeared to be in a dream state or delirium. He kept his eyes closed and grunted like an animal. At that time his temperature was normal, and this without the cerebrospinal fluid findings, at first suggested an hysterical state. He later became distinctly unclear and disoriented. All these irritative symptoms preceded the lethargic state.

4. The substupor or lethargy did not appear until the neurologic signs became marked, more particularly eye signs, diminished knee reflexes and bladder symptoms. From this low grade stupor he could be aroused up to the last day or two, when the terminal coma set in.

Case 4.—*History.*—H. H., a white man, a Russian Jew, aged 34, married, a sign painter, whose history was unknown to the wife, was negative according to the patient. He was born and educated in Russia. He had been in this country ten years, and spoke English unusually well. His best wage was forty dollars a month. His previous medical history was unimportant except for appendectomy about June, 1918, and influenza in the winter of 1918-1919.

Make-Up of the Patient.—He was married in August, 1919. He had neurotic tendencies before and after marriage. He was known to his wife only three months before marriage. Then and shortly after he expressed ideas of impotency; he was frequently unable to accomplish orgasm. Several months before his present illness he saw several physicians in regard to this condition.

Present Illness.—The patient was first seen on Feb. 10, 1920. For a few weeks before he had complained of so-called "neuritis," with pains at the back of the neck, and at the outer aspect of the right arm. Repeated examinations revealed nothing important, except an enlarged turbinate; ethmoidectomy was performed and later the sphenopalatine ganglion was injected with alcohol; following this the patient seemed to improve for a short time, but his former symptoms returned, and when first seen he complained of various pains localized in the right shoulder, the right mastoid region, at the vertex of the cranium and in other places. He described the pain as being sharp and like that of a hammer beating him every few minutes.

Examination.—Feb. 10, 1920: A neurologic examination from an organic point of view appeared to be entirely negative, except for an exaggeration of all the deep tendon reflexes. There was a marked glove and upper arm analgesia and anesthesia. This was less marked in the lower extremities. Hemihyperalgesia of the right face, arm and trunk appeared at a later interval. The cranial nerves were normal.. Vibratory and muscle tendon sense and electrical reactions were normal.

Mental Picture.—The patient was groaning and talking in a whispered voice. On being questioned, he said he did not realize that he was whispering. When his attention was distracted all evidence of pain seemed to disappear. On compression of the eyeballs, the patient said he saw various colors, flowers, trees, "now a castle, now a lighthouse with the sun as though it were going down."

On February 14 and 15 his condition was unchanged. He was put under hypnosis. During the second hypnotic state he described a journey through Arabia. He stoutly denied all memory of falling to sleep. During the waking state he talked spontaneously about his sexual impotence, and showed lack of insight when he could not be convinced that he could have a child (his wife was then pregnant) and still be impotent. From February 10 to 15 his temperature was usually normal, occasionally being slightly subnormal, and on one occasion it was 99 F.

Apomorphin had only a slight effect in doses of 1/10 grain three times daily. This was directed to jerking movements of the abdomen which seemed to be within the patient's control on and after February 13. On the 16th his temperature began to rise gradually. A spinal puncture was performed with the following results: cell count, 20; colloidal gold, curve flat; globulin, negative, and Wassermann reaction negative. On the same day he began to develop visual and kinesthetic hallucinations. He thought his bed was wired with electricity and imagined that chickens were being hatched under his arm pits

and under his pillow. He also developed suspicious tendencies against a brother who was summoned from New York.

On February 20, Dr. Albert M. Barrett and Dr. Carl D. Camp were called in consultation from Ann Arbor. At that time both knee reflexes were doubtfully obtained, there was a noticeable horizontal nystagmus, a slight ptosis of both lids and a distinct bilateral weakness of the sixth nerve. The nurse reported that he was constantly masturbating or manipulating the genitalia. The nurse also reported that the patient rehearsed in detail to his brother all that was said at his bedside by the physicians.

His case was looked on as one of so-called lethargic encephalitis.

Later Clinical Course.—Feb. 20, 1920, nasal examination was practically negative, except that there was a slight erosion of the anterior portion of the septum with dried bloody secretion. There was a slight deflection of the septum; the left nasal cavity was not obstructed. The blood count revealed 13,600 white cells. The temperature remained constantly elevated, ranging from 99 to 102 F., but averaging about 100 until April 20, when it became slightly subnormal and remained so for a week. It then returned to normal and remained so until the time of his discharge, May 23, 1920.

The jerking movements of the abdominal muscles persisted until about March 15, when they gradually began to lessen in intensity though they never disappeared entirely. At no time were there any bladder symptoms.

Mental Picture.—For a short time after admission to Harper Hospital, Feb. 16, 1920, the condition was much the same as that described above: He was restless, picked at the bed clothes, had hallucinations, was disoriented and at times incoherent. This condition gradually disappeared until about March 1, when a substuporous or so-called lethargic state appeared and continued for about a week. He could, however, at all times be aroused sufficiently to answer questions.

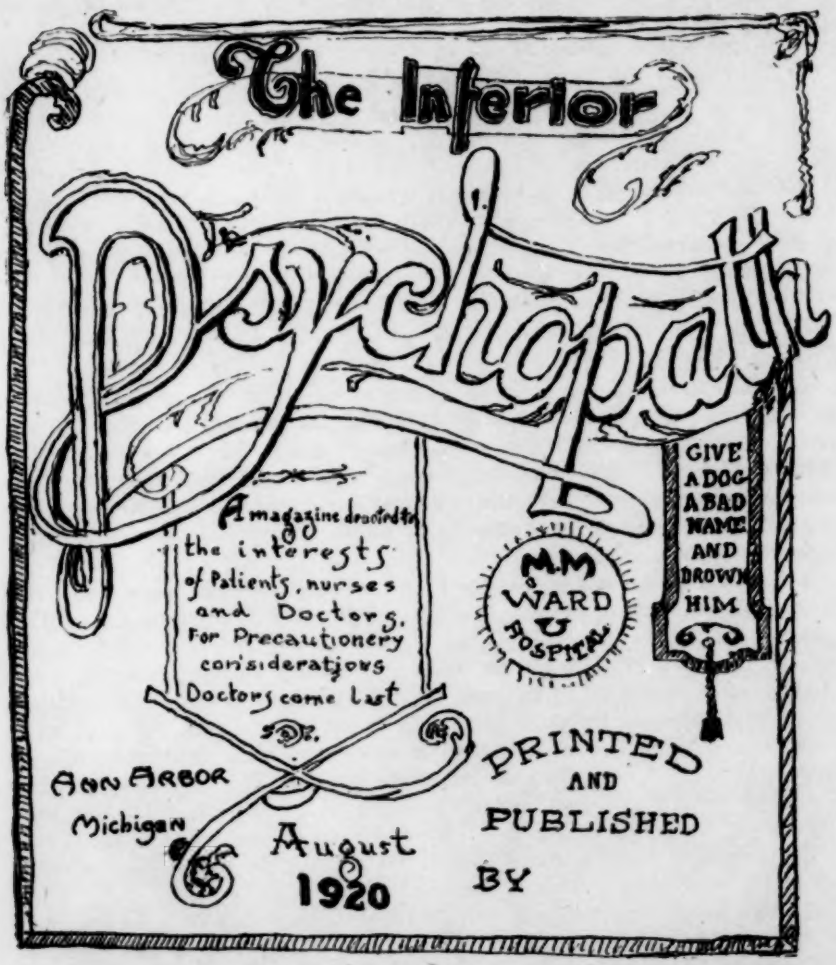
On or about the middle of April, two months after the acute onset, the patient began to have insight into his condition, but there was a distinct amnesia for the period of irritation or delirum and for the period of substupor. On April 18, he wrote to Dr. Mc.: "Having emerged from the world of shadows—a world all of its own, with its own laws, where the glimpses of reality that penetrate the mind serve only as material to draw the most weird pictures, and being now on the road to recovery, I take this opportunity to thank you . . . for calling on me at the hospital . . . although I don't remember it . . . people tell me about it . . . and last, but not least, I thank you for giving me Dr. J.—to whom I have so much to be thankful for."

The period of amnesia dated back to the time of the first lumbar puncture before his admission to Harper Hospital.

Though insight had returned, and the patient's mind was clear, he continued to show marked psychoneurotic tendencies. He seemed to delight in localizing the areas in which he stated he still had pain. He would point to one knee and say "I have pain here; but that is not all. My pain must be symmetrical, and so I have pain here," pointing to the same spot on the opposite knee. He would do this for almost all portions of the body. He would frequently speculate and dilate on his experiences and symptoms during his illness, making it appear at times that his amnesia was not complete. Whenever any mention was made about its being almost time for his discharge from the hospital, he would become querulant and complaining. He was seen once or twice in the outpatient department after his discharge, and on each occasion showed a lack of initiative, dilated on his various bodily weaknesses and

begged to be readmitted. He returned to work but only for a few days. He seemed helpless at home and continued to complain until he was finally admitted to the University Hospital at Ann Arbor on an order of the court. He is still a patient at that hospital.

That the psychopathic tendencies exhibited before his illness have not disappeared with convalescence is evident from the following interesting katamnesis. While awaiting the order from the court for his admission as a public


The Inferior
Psychopath
A magazine devoted to the interests of Patients, nurses and Doctors. For Precautionery considerations Doctors come last
GIVE A DOG A BAD NAME AND DROWN HIM
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Cover page of "magazine" written by the patient in Case 4. This cover was designed and drawn by the patient after recovery.

patient to the University Hospital, it was necessary to detain him in the Psychopathic Ward at the Detroit City Receiving Hospital. By some chance he saw his chart or heard notes read from it and caught up the words "psychopathic inferiority" relative to his own case. Under date of Sept. 6, 1920, he wrote to Dr. Mc. a detailed letter, in the form of "A Magazine Devoted to the Interests of Patients, Nurses and Doctors. For Precautionary Consid-

erations Doctors Come Last." It was painstakingly printed by hand in pen and ink script with an illustrated cover and three pages of text. An exact reproduction of the cover is shown on page 158. A few excerpts from the "magazine" may be of interest. Evidently he no longer thanks Dr. Mc. for giving him Dr. J.

"Dear Dr. Mc.:

"Now, that I have several painless hours every day, and can sit at the writing table like a man, I wish to let you know that my wife gave birth to a baby girl.

"She is now nine weeks old . . . very beautiful and is an exact copy of me (there is no discrepancy in it, for I was nice looking when I was a baby) yours sincerely, H— H—, the individually psychopathically inferior following encephalitis lethargica.

"No, Doctor, I did not give myself deliberately such a name, it is a bull conceived in an unfortunate hour by Dr. J—. With this name, he sent me for a few days to the Receiving Hospital when they locked me up with insane criminals. When I asked the nurse . . . for a pillow . . . she told me to cut it out, and pointing to the history of my case, . . . she said that I am insane . . . and simulating pain because I want everybody to serve me . . . although she did not say it, but what she meant was probably that I am an egomaniac or afflicted with mania grandiosa. . . . Since I awoke from that long sleep, I have not spoken one word which would justify such a diagnosis. I cannot have anything against the deductions of the science of psychiatry, and I would not worry so much and it would not be so painful if some one else did it, but Dr. J— who has done for me so much, who has literally wrested me from the jaws of death, of him I did not expect this. And then I don't understand what it means, 'Inferior psychopath;' this implies the existence of 'superior psychopaths.' What do they look like?

"I know I was irrational during the first period of my illness; I don't know what the future holds for me; but at present am S a n e. Or is it the fact that the cause of the pain is in the brain that made him call me that name? then why inferior? and would not 'Pathologic brain' or some other term do?"

This letter apparently indicates that the patient's condition is approximately what it was before his illness: he is an odd type with neurotic traits thrown into stronger relief by the physical after-effects of his earlier toxic state.

Summary of the Mental Picture of Case 4:

1. Russian Jew, fairly well educated, of distinct neurotic type.
2. Fears of sexual impotency before and after marriage.
3. Toxic character of illness masked in its early stages by what appeared to be purely hysterical symptoms, including anesthesias, analgesias, easy suggestibility with successful hypnosis and other symptoms, as well as aphonia.
4. After the toxic character of the illness was manifest there appeared symptoms of irritation, mental unclearness and hallucinations; ideas of influence, delusions, disorientation, masturbatory tendencies, picking at the bed clothes and other symptoms.
5. Still later appeared the so-called lethargic, or substuporous stage from which the patient could be aroused sufficiently to answer questions.
6. Finally the amnesia, partially questionable, and the tendency to defense mechanisms seen in his letters. The so-called "magazine" illustrates this well, and even suggests hypomanic and dementia praecox features.

In reviewing the four cases just reported there seem to be two outstanding features of the disease, one not limited to the disease entity before us, and the other possibly generally characteristic of the

disorder, although not strictly limited to it. Without attempting to relate these features to the pathology of encephalitis, and looking at the subject from a clinical point of view, these two features seem to be: one, the symptoms of the irritative stage, and the other the symptoms of the so-called lethargic, substuporous or low grade stupor stage. That of the irritative stage is less characteristic and is a feature so frequently found in what is popularly styled "nervousness," "maniacal states," or "delirium" (dependent on the degree of irritation) of the various toxic psychoses. The most important irritative symptoms in the four cases just reported may be recapitulated in the order in which they present themselves:

Case 1: During illness there were marked emotional instability, unclearness or mental confusion, temporary loss of speech and amnesia for the delirium. After recovery emotional instability without deterioration appeared.

Cases 2: Terrifying dreams, insomnia, distractibility, mild kinesthetic hallucinations, failing powers of concentration, a tendency for odd numbers and letters to fix themselves in his consciousness, and a mild degree of euphoria were present. He manipulated the genitals (this may be regarded as purely a physical, rather than a psychic irritation). After recovery there were no irritative symptoms. Case 3: The patient was in a dream-state or delirium and made grunting noises like those of an animal. His eyes were closed (possibly photo sensitivity), and he gave evidence of unclearness and disorientation, an incoherent stream of thought, and delusions of a mild persecutory character.

As for the substuporous stage, all of the patients except the patient in Case 1 showed some degree of lethargy or "emotional paralysis." [1a] All three patients could be aroused from this condition, with the exception of the patient in Case 4 during his terminal coma; all three showed some degree of so-called parkinsonian or ironed-out facies, and all had an amnesia for both the irritative and lethargic periods, less marked for the lethargic period as this projected itself in two of the cases, in milder form in the period of convalescence or recovery.

The patient in Case 2 four months after convalescence temporarily dozed off into a brief drowsiness during his work.

Two other interesting psychiatric elements present themselves in all four cases. 1. The patient either had a tainted heredity or had shown marked psychoneurotic tendencies before his illness. In Case 1 there was insanity in the ancestry and the patient was being reared in the atmosphere of divorce and other family difficulties. The patient in Case 2 was of a high-strung disposition and was temperamental.

1a. Wechsler, S.: The Symptoms of Epidemic Encephalitis Structurally and Functionally Considered, New York M. J. **112**:175 (Aug. 7) 1920.

In Case 3 there was a history of petit mal epilepsy, with hysterical symptoms, and the patient in Case 4 was a neurotic of the nth degree with sexual obsessions, suggestible to the point of hypnotism and a more or less helpless type of individual.

2. The three patients who recovered showed some residuals of the acute illness long after convalescence set in. One was emotionally unstable, another had brief periods of drowsiness and the previous tendencies of the fourth were highly accentuated. He imagined that he was the victim of ill-directed medical classification, and spent his time in writing letters not unlike those of a hypomaniac. He is rapidly becoming institutionalized and it is questionable whether he will ever again properly maintain himself and his family.

This last point, as far as the variable symptoms of the disease are concerned, is probably the most important. In short, the mental picture of the irritative stage as well as that of the post-convalescent stage, with residuals, is determined by the individuality of each patient. Consequently, no two cases are alike from the psychiatric point of view, particularly when analyzed relative to the irritative symptoms. The situation here is much the same as it is in general paralysis, dementia praecox and the other psychoses. Each patient has his own background on which the changing features of his acute illness are thrown and thus make the psychic element unique.

REVIEW OF LITERATURE

Although epidemic encephalitis, as such, has been accorded much attention in the literature, particularly during the past year, yet little, beyond casual mention in the better reviews and the occasional report of isolated cases, has been contributed on the more essentially psychiatric phase of this disorder, notwithstanding the undoubted importance of such an analysis.

On the peculiar lethargy or apathy so characteristic of the disease and responsible for its original designation, lethargic encephalitis, by Economo,[2] in 1917, much comment has been made. This state has been variously characterized by different observers as cerebral torpor (Cruchet [3]), or somnolence (Netter and Sainton [4]), or stupor with normal rationality (Tilney and Howe [5]), and seems to have varied, in degree, from mere listlessness or mental inertia, to frank coma,

2. Economo: Verein f. Psychiat. u. Neurol. in Wien., April 17, 1917.

3. Cruchet, R.: Bordelaise Conception of Encephalitis Lethargica, New York M. J. **112**:173 (Aug. 7) 1920.

4. Netter, A., and Sainton: L'encephalite lethargique, Presse méd. **26**:487 (Sept. 23) 1918.

5. Tilney, F., and Howe, H. S.: Epidemic Encephalitis, New York, Paul B. Hoeber, 1920, pp. 85, 20, 60, 50, 96.

with catatonic features. The condition, on critical analysis, seems not to have been coma or even true stupor as it appears to have been generally possible, on proper stimulation, to recall the patient to objective consciousness for brief periods, during which, beyond in some cases a certain retardation or lengthened reaction time, there was but little evidence of mental disturbance or impairment; although, as suggested by Burr,[6] it is questionable whether in the absence of external stimuli there was actually any mediation of mental processes or any emotional tone. Wechsler [1a] suggests resemblance to an emotional paralysis, and Alexander and Allen [7] include reference to it as an emotional stupor as does also Kennedy,[8] who has characterized such cases as intellectually keen but emotionally stupid. The term pseudostupor, as suggested by Burr,[6] seems well in point although it is questionable whether some such designation as substupor might not be more suitable.

Wechsler [1a] suggests that conceivably this state may have a thalamic basis and mentions the rather doubtful possibility of basic pituitary or pineal influence. On the whole, however, as noted by Burr [6] and Tilney and Howe,[5] the condition seems more probably but a manifestation of the generalizing nature of the condition, as is borne out also on consideration of its basic pathology (Hammes and McKinley [9]). Russell [10] thinks that the somnolence depends entirely on a lesion in the posterior part of the pons near the aqueduct of Sylvius, thus interfering with the flow of the cerebral fluid in the cerebral hemisphere to the spinal canal and resulting in an acute hydrocephalus.

The so-called catatonic features—catalepsy, flexibilitas cerea, parkinsonian facies and the other features—have been reported by most observers, notably by Bond,[11] Hammes and McKinley,[9] Wilson,[12] Hall,[13] Kennedy,[8] Batten and Still,[14] Tilney and Riley,[15] Tilney and Howe,[5]

6. Burr, C. W.: Case Report, Trans. Philadelphia Neurol. Soc., Feb. 27, 1920; Arch. Neurol. Psychiat. **4**:105 (July) 1920.

7. Alexander, M. E., and Allen, H. E.: Lethargic Encephalitis, Arch. Neurol. & Psychiat. **3**:485 (May) 1920.

8. Kennedy, F.: Epidemic Encephalitis with Stupor, Med. Rec. **95**:631 (April 19) 1918.

9. Hammes, E. M., and McKinley, J. C.: Lethargic Encephalitis, Arch. Int. Med. **26**:60 (July) 1920.

10. Russell, C. K.: Study of Epidemic Encephalitis Based on Seventeen Cases with Two Necropsies, Canadian M. J. **10**:696 (Aug.) 1920.

11. Bond, E. D.: Epidemic Encephalitis and Katatonic Symptoms, Am. J. Insan. **76**:281 (Jan.) 1920.

12. Wilson, S. A. K.: Epidemic Encephalitis, Lancet **2**:7 (July 6) 1918.

13. Hall, A. J.: Epidemic Encephalitis, Brit. M. J. **2**:461 (Oct. 26) 1918.

14. Batten, F. E., and Still, G. F.: Epidemic Stupor in Children, Lancet **1**:636 (May 4) 1918.

15. Tilney, F., and Riley, H. A.: Epidemic Encephalitis, Neurol. Bull. **2**:106 (March) 1919.

and Bassoe.[16] The exact mechanism concerned in the production of the muscular hypertonus constituting this feature has apparently not yet been definitely determined. Thus Tilney and Howe [5] seem to feel that it is due essentially to extrapalliospinal involvement, while Wechsler [1a] questions whether this sign can be explained wholly on the basis of extrapyramidal involvement and mentions the possibility of a true cerebral condition, an interference with cerebral functioning, with a resultant setting free of uninhibited unconscious activity, as found in dissociative epilepsy.

The possibility of the existence of a definite or characteristic psychosis is deprecated by Wechsler, who feels that findings suggestive of such a state are but manifestations of an underlying infection or toxic delirium syndrome. Tilney and Howe, in the inclusion of epileptomaniac and acute psychotic types in their clinical classification, seem to bear out this point of view, as do also observations by Wilson.[12] Cases studied by Hammes and McKinley [9] showing delusions, hallucinations and temporary disorientation, and by Cruchet,[3] showing disorientation and paraphasic features, may be construed on the same basis.

On the other hand, Climenko [17] reports a case of definite Korsakow psychosis and Leiner,[18] Hammes and McKinley, and Wilson remark the occurrence of Korsakow-like syndromes.

Leiner mentions two apparently frank cases of manic depressive psychosis, one of the cyclothymic type. Climenko also indicates this possibility, as an end point to the characteristic euphoria which he frequently found to precede the acute phase of the disease. This euphoria, he finds, shows a definite tendency to become a hypomania, as the disease progresses which, in turn, may become a truly manic condition. He further indicates the possibility of the development, in some subjects, of an excited-depression as opposed to a hypomania. Euphoria has been noted in addition, by Tilney and Howe,[5] Schwartz,[19] and House.[20]

Wilson [2] reports a case in which the patient developed a typical witzelsucht.

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16. Bassoe, P.: Epidemic Encephalitis (Nonna), J. A. M. A. **72**:871 (April 5) 1919.

17. Climenko, H.: Encephalitis Lethargica, New York M. J. **111**:531 (March 27) 1920.

18. Leiner, J. H.: Encephalitis Lethargica, New York M. J. **111**:179 (March 27) 1920.

19. Schwartz, S.: Encephalitis Lethargica, New York M. J. **112**:6 (Aug. 7) 1920.

20. House, W.: Epidemic (Lethargic) Encephalitis, J. A. M. A. **74**:372 (Feb. 7) 1920.

The available literature apparently indicates that psychiatrically considered the clinical picture in epidemic encephalitis is still somewhat indefinite. Nevertheless, there appears to have been definitely determined the characteristic initial pseudostupor or substupor (which may be preceded by a euphoric phase) distinguished by marked depression of the emotional field, often with catatonic features. In addition, as a function of the generalizing character of the infection and the reaction to it, there is evidence strongly suggestive of infection, toxic delirium or toxicorganic psychosis, which in some cases appears to simulate strikingly certain other common, noninfectious psychoses.

In the latter regard, although no specific mention has been made, there is no reason to suppose that a latent psychosis may not be precipitated as a result of the infection, as has been determined in the case of infectious processes, notably epidemic influenza.

CONCLUSIONS

1. The psychiatric phase of epidemic encephalitis has not received the same study and attention as has the neurologic aspect.

2. Looking at the disease from the psychiatric aspect, there seem to be two stages—the irritative stage in which the mental picture is not unlike that of other noninfectious psychoses, and the period of "low grade" stupor, lethargy or, better still, the substuporous period. The former may be marked by euphoria, hypomania, depression, hallucinations, paranoid stages, or even the Korsakoff syndrome, and frequently there are features that simulate those of severe hysterical excitement. The latter is rather an extreme apathy from which the patient may be roused; there is, as it were, a paralysis of the emotional field, frequently accompanied by masklike or parkinsonian facies. This apathy or stupor in some cases amounts almost to a catalepsy, but it should be remembered that it is possible, except in the terminal stage, to rouse the patient.

3. The keynote, if we may use this figure, is that the mental condition is variable, this condition being an infectious-toxic one, the whole dominated at some time by an apathy or substupor in which the personality is fairly well preserved but blurred by torpor.

4. The authors' four cases bring out these interesting features of the disorder:

(*a*) The symptoms during the irritative stage reflect the patient's make-up previous to his infection.

(*b*) There is an amnesia for the irritative period. In these two respects, the disease is characteristic of a toxic delirium.

(*c*) Each patient experienced the substuporous stage except the first, a child of 9. This patient was not seen until some time after the onset.

(*d*) There are mental residuals in all patients that recovered, either similar to those noticed in the person before his illness, or else highly aggravated. These may gradually pass away, or become progressively more pronounced. The last result is true of Cases 1 and 4.

5. It may not be dogmatically maintained that the disease can be diagnosed from the psychiatric picture; the substuporous stage, however, is strongly suggestive of the disorder, and when found in conjunction with spasmodic twitchings, lost knee reflexes, bladder disturbances, cranial nerve palsies and other symptoms is almost if not truly diagnostic.[21]

21. Since the manuscript for this paper was submitted, two articles have appeared: one, in the November issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, by L. Archambault. He lays emphasis on the generalizing nature of the infection and speaks of one case in which there was a paresis-like picture. The other is by I. Abrahamson: "Mental Disturbances in Lethargic Encephalitis," J. Nerv. & Ment. Dis. **7**:193 (Sept.) 1920. He also speaks of the generalizing nature of the intoxication and of the Korsakoff-like pictures in the disease.

A CASE OF ALTERNATING HEMIPLEGIA (MILLARD-GUBLER), WITH REMARKS ON THE PATHS OF THE PUPILLO-DILATOR AND VESTIBULO-OCULAR FIBERS IN THE BRAIN STEM

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Sharply defined anatomic lesions of the brain stem have recently acquired a new interest in so far as they are now able to furnish us exact information as to the course of those fibers which come from the upper part of the mesencephalon and extend to the centrum ciliospinale. Irritation of these fibers produces psychic as well as reflex dilatation of the pupils. Information is also furnished regarding the path of those other fibers that transmit the irritation from the vestibular apparatus to the oculopontile nuclei. The following case is of interest in this connection.

REPORT OF A CASE

History.—Felipe de Ll., 31 years old, an engineer, complained Oct. 23, 1918, of reduction of visual acuity. He had always been healthy and strong. There were no family diseases. He denied syphilitic infection. When he was 11 years old, he fell from a height of 3 meters, on his cranium; as a consequence of this accident, there was a small nonadherent cicatrix of the skin in the right parietal region.

Since January, 1918, he had observed indications of metamorphopsia in both eyes, and reduction of his visual power. On June 21, 1918, he suddenly became ill, with symptoms of a left-sided hemiplegia, including the left side of his face, his arm, trunk and leg; at the beginning of this attack he was unconscious for several hours.

Examination.—Examination Oct. 31, 1918, revealed a pale, young, strong, well-nourished man, who complained of headache in the region of the right fissura centralis. The urine contained a trace of albumin (0.08 0/00), $\frac{1.200}{1.019}$ no sugar and no casts. The Wassermann reaction of the blood was negative. Percussion of the cranium did not produce pain. There was no Romberg sign or symptoms from the cerebellum. The tendon reflexes were normal on the right side, but seemed to be a little diminished on the left side. There were no indications of contracture and no Babinski reflex. The muscular force of the left arm and left leg were undoubtedly reduced, as well as the sensibility. The pupils were equal in both eyes and of medium width; they reacted well to light and accommodation; there were no anisocoria, diplopia, nystagmus, or reduction of the eye movements. Ophthalmoscopically the disks appeared blurred; they showed no prominence, and the entire fundus was covered with small white spots as in albuminuric neuroretinitis. The visual field showed a slight reduction of color sensation in the temporal half of the left eye, being otherwise normal.

Dec. 23, 1919: The ophthalmoscopic alterations had receded somewhat; otherwise the status was exactly the same as at the time of the previous examination. Examinations of the blood showed negative Wassermann reactions; and many examinations of the urine disclosed no evidence of nephritis. An examination of the cerebrospinal fluid showed a negative Wassermann reaction and a positive globulin reaction (Noguchi), with slight increase in quantity. The original diagnosis of neuroretinitis albuminuria could no longer be maintained. An examination of the vestibular apparatus by Dr. Fernandez Soto showed:

1. Slight reduction of the hearing of the right ear on account of cicatricial otitis (the lesion was caused by an insect in his youth).

2. Both acustici proved to be normal.

3. No spontaneous nystagmus, but spontaneous past pointing of the left arm toward the right. Examination on the turning chair revealed: in different investigations, slight reduction of the amplitude and duration (18 seconds) of the normal nystagmus from the horizontal canals, normal past pointing and vertigo. Ear stimulation with warm water, for the examination of the vertical canals, revealed a complete absence of nystagmus, past pointing, and vertigo; on changing the position of the head (horizontal canals) a reduced nystagmus, past pointing and vertigo appeared. There existed, therefore, a complete inexcitability of the vertical semicircular canals, and hypo-excitability of the horizontal canals. The patient showed no signs of labyrinthine disease (spontaneous noises, vertigo or deafness).

Jan. 21, 1920: On the entire left side of the patient's body (face, arm and leg) a well-marked thermoanesthesia appeared; a few days before he had an attack of dysarthria lasting for some minutes; there was no Babinski reflex.

March 8, 1920: While in the toilet, he suddenly lost consciousness and fell to the floor. He showed a flaccid paralysis of the left arm and left leg (but not of the face) and anesthesia of the entire left half of the body (including the face). The anesthesia could be observed especially well during sleep; one could prick him with a needle on the left side to the point of drawing blood and he showed no reflex defensive movement and did not waken; the reflex defensive movement was marked on the right side. Insensibility extended exactly to the middle of the nose and was most marked when the patient was awake, for temperature and pain and less marked for touch.

March 10, 1920: The patient complained of violent pains and pronounced hyperesthesia in the whole region of the right trigeminus.

March 18, 1920: The patient showed a total peripheral paralysis of the right facialis.

April 17, 1920: Ophthalmoscopically the patient showed in both eyes a well marked choked disk of 3 to 4 diopters. The visual field was normal. The trigeminal pains had disappeared. Vomiting, vertigo, cerebellar symptoms and anisocoria were not present. He had only paralysis of the right facialis and paresis and hypesthesia of the left arm and leg.

April 27, 1920: During the four days before death the patient again suffered pains in the right trigeminus, which increased to such violence that he attempted suicide. On April 27 he felt much better; he arose, and after taking a short walk, he fell, began to perspire copiously, and died within a few minutes with Cheyne-Stokes respiration. Necropsy examination was refused.

Summary.—A young, strong, nonsyphilitic man, in whom no signs of renal diseases could be traced with certainty, began to complain of metamorphopsia and loss of vision, and showed ophthalmoscopically the appearance of neuroretinitis albuminuria. A complete left-sided hemiplegia (face, arm, leg) receded, leaving only a marked anesthesia of the whole left side, more pronounced for temperature and pain. There were no signs of contracture, Babinski reflex, or exaggeration of the tendon reflexes. An examination of the ear failed to disclose disease but showed a complete lack of the function of the vertical semicircular canals and a slight reduction of the function of the horizontal semicircular canals. The patient did not have the signs of brain tumor. As time passed complete right-sided paralysis of the facialis and a marked hyperesthesia of the right trigeminus with spontaneous pains developed, added to the left-sided slight disturbance of motility and strong alteration of sensibility. The neuroretinitis albuminuria developed into a typical bilateral choked disk, and the patient died suddenly with symptoms of vagus death.

Necropsy having been refused, cause and localization of the anatomic lesion could not be ascertained with the desired exactness, but we may deduce the following:

The lack of casts in the urine, its normal specific weight and its normal quantity tend to show that a chronic renal malady did not underlie the disease. We have many observations in the earlier ophthalmologic literature showing that the most experienced ophthalmologists pronounced the diagnosis of "nephritis chronica" on account of the fundus picture, while the necropsy failed to discover any disorder of the kidneys but proved the presence of a brain tumor. The whole course of the disease, the developing of a typical choked disk, the positive globulin reaction in the cerebrospinal fluid, the development of a crossed hemiplegia and the sudden death under the symptoms of Cheyne-Stokes' respiration, indicate that we were dealing with a slowly advancing process in the brain stem, probably a tumor, or perhaps a tuberculum.

With greater exactness we can deduce the location of the lesion. The reduction of the sensibility in the whole left side of the body (face, arm and leg) shows an interruption of the lateral fillet between its crossing in the medulla oblongata and its termination in the thalamus opticus; it further shows that this lesion must be located on the right side of the brain stem and higher up than the entrance of the fibers of the trigeminus after their crossing, i. e., in the middle third of the pons. The right-sided peripheral paralysis of the facialis and the right-sided hyperesthesia of the trigeminus indicate, furthermore, a lesion on the right side of the middle part of the pons. The fact that the sensation of pain and temperature alone was completely abolished on the left side of the body and that superficial sensibility was only reduced, together with a peripheral palsy of the facialis without disturbance of the abducens; also the normal functioning of both acustici and the dissociated lesion of the vestibular fibers, and the flaccid character of the left-sided hemiplegia, show that there was a sharply defined anatomic lesion on the right side of the brain stem situated in the upper and external part, limited ventrally by the pontile nuclei, laterally by the vestibular nerve and mesially by the abducens and the mesial fillet; it did not ascend to the floor of the fourth ventricle, because the nucleus of the abducens and the fasciculus longitudinalis posterior were not altered in their function. The lesion included the descending root of the trigeminus (spontaneous pains on the right side

of the face) and destroyed the lateral fillet, the right pyramidal fibers dispersed between the pontile nuclei and the exit of the right facialis. On the right side the function of the facialis and trigeminus were abolished; on the left side, the greater part of the sensibility and the motility of the arm and leg were abolished. This syndrome has long been known as hemiplegia alternans (type of Millard-Gubler), and it would scarcely be worth while reporting it, were it not for three special points.

ABSENCE OF DEVELOPMENT OF CONTRACTURE

Notwithstanding that the patient showed twice, with an interval of about two years, a disturbance of the right pyramid, the paralysis always had a flaccid character and no signs of contracture developed. An interruption of the supranuclear motor path is followed as a rule by a spastic paralysis, and even if it completely recedes, an exaggeration of the corresponding tendon reflexes remains, and a positive Babinski reflex develops. The signs of contracture develop at the earliest after fourteen days, but as a rule from one to three months after the attack, and they fail to appear only in the rarest exceptions. Explanation of the developing of the cerebral contracture has been attempted, and careful investigations have shown the interruption of the pyramidal tract and the integrity of the posterior roots to be the essential conditions. Both conditions were present in my case, and nevertheless the reflexes of the left side were rather diminished. Of the different theories, that of van Gehuchten is best suited to the present case; it holds that the contracture is caused by the continuance of the functioning of those fibers which connect the pontile nuclei with the cerebellum (fibrae cortico-ponto-cerebello-spinales) through the middle cerebellar pedunculus.

LACK OF LESION OF THE PUPILLO-DILATING FIBERS

During the whole course of the disease I paid special attention to the observation of the pupillary reaction in order not to overlook the appearance of anisocoria. We know from many observations that a lesion of the brain stem is sometimes followed by a homolateral miosis, and therefore lesions of the brain stem are apt to illustrate to us the exact topographical course of the pupillary fibers. If we wish to determine their situation in the brain stem, we must look for the following points: (*a*) height and latitude of the lesions, and (*b*) crossing of the fibers.

(a) During the entire course of the disease both of the patient's pupils were equal and of medium size. This agrees with other symptoms that indicate a lesion in the tegmentum of the pons not having reached the floor of the fourth ventricle. Déjerine became convinced, through his anatomic investigations, that the sympathetic fibers run through

the upper layers of the substantia reticularis, medially of the root of the trigeminus and laterally of the middle of the brain stem. In a previous article I expressed the opinion [1] that the sympathetic fibers are responsible for the pupillo-dilatation and that they run down from the mesencephalon to the centrum cilio-spinale, together with the fasciculus longitudinalis posterior which is situated just on top of the substantia reticularis. Spiller [2] gave some further instances showing that the lesion of the brain stem can be followed by a homolateral miosis, and in his cases the sympathetic fibers have been interrupted by foci of destructive nature. In consideration of this point, tumors like gliomas which entangle the fibers are less apt to give information than tuberculous foci that destroy them. As we have not yet many observations of lesions of the brain stem which especially consider the localization of the pupillo-dilating fibers, it may be well to cite here, two further examples illustrative of the subject. F. Raymond [3] gives an exact description of a case closely resembling my own: Paralysis of the facialis and of the abducens on the left side, and spastic paresis of the right arm, trunk and leg, together with cessation of all sensibility on the right side of the body (face, arms, trunk and leg) occurred; the patient showed no anisocoria. Another patient [4] who had, on the right side, hemiparesis and hypesthesia of the arm, trunk and leg, and on the left side a paralysis of the facialis, abducens and masseter, together with anesthesia of the left side of the face, had a miosis without alteration of the pupillary reaction on the left side. The fact that the masseter was paralyzed shows that the lesion extended higher up in the level and extended more medially than the previously reported case and my own.

We may therefore conclude that the lesion of the brain stem will be accompanied by a homolateral miosis only when it reaches the highest and medial part of the substantia reticularis.

(b) Most authors believe, with me, that in man the sympathetic fibers do not undergo a crossing between the mesencephalon and the centrum cilio-spinale; some question that they undergo any crossing at all.

In favor of the first opinion that the pupillo-dilation fibers running down from the mesencephalon to the centrum cilio-spinale (which is

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1. Lutz, A.: The Light Pupillary Reflex, Its Path and Its Abolition Called Immobility of the Pupil to the Light Reflex, and Report of a Case of Unilateral Argyll Robertson Pupil in which Consensual Reaction Existed in Both Eyes, Arch. Ophth. **47**:266 and 370, 1918.

2. Spiller: The Oculo-Pupillary Fibers of the Sympathetic System, Am. J. M. Sc. **100**:325, 1920.

3. Maladies du systeme nerveux, 1900, p. 63.

4. Maladies du systeme nerveux, 1903, p. 436.

the homologue of the motor cells of the anterior horns for the pyramidal system) remain uncrossed on the brain stem, are the above named reports on anotomic lesions, as well as the experiments of Levinsohn.[5] Levinsohn cut the brain stem and the medulla-spinalis cervicalis in rabbits on one side and observed that this lesion was followed by a homolateral miosis. This miosis was always homolateral and not pronounced, but unmistakable, when he made the cut through the medial parts of the brain stem above the trigeminal entrance. Miosis was stronger and mostly homolateral, but sometimes also in a minor degree heterolateral after he had made the cut below the entrance and more lateral (descending roots of the trigeminus) miosis was never accompanied by a reflex immobility of the pupil. This developed only when he made the cut at a height between anterior and posterior corpora-quadrigemina. Levinsohn explained his experimental miosis by way of irritation of the pupillo-constrictor fibers, instead of by way of paralysis of the pupillo-dilating fibers. Therefore Levinsohn found in rabbits, as did Karplus and Kreidl[6] in cats, that each half of the cervical cord conducts impulses from the brain stem to both cervical sympathetic nerves. In man, however, all our experience tends to show that the miosis is homolateral with a lesion of the brain stem. This shows us that the important experiments of Karplus and Kreidl made on cats and apes with regard to the subthalamic dilation center and its connection cannot be applied unreservedly to human pathology.

The question of crossing is more difficult to decide in the parts of the pedunculus situated higher up because of the lesion occurring at the same time in the fibers of the oculomotorius responsible for pupillo-constriction. For the following reasons we may conclude that they do not undergo crossing below the anterior quadrigemina: Brissaud[7] remarks that an ophthalmoplegia externa pura is as a rule accompanied by a miotic pupil without alterations of the reactions. Cases of unilateral Argyll Robertson pupil, such as are reported by Tanzi,[8] Salomonsohn,[9] and myself,[10] show miosis. The results of Levinsohn's experiments with rabbits, and cases of hemoplegia reported by Spiller, and the case of unilateral thalamic lesion of Ingham, referred to by Spiller in the same article, apparently point to this conclusion.

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5. Beiträge zur Physiologie des Pupillarreflexes, Von Gräefes Arch. f. Ophth. **59**:191, 1904.

6. Karplus and Kreidl: Arch. f. d. ges. Physiol. **129**:138, 1909; 1910, p. 401; 1912, p. 109.

7. Maladies du systeme nerveux, 1895.

8. Riv. di patol. nerv., 1899.

9. Von Gräefe's Arch. f. Ophth. **54**:211, 1902.

10. Arch. of Ophth. **47**:266, 1918.

If we deduce from the foregoing that the pupillo-dilation fibers do not cross, in man, between corpus quadrigeminum anterius and centrum cilio-spinale, we are forced to search for the crossing higher up. For it is a general anatomico-physiologic law that every efferent motor system undergoes a crossing in its supranuclear path. We must further remember that there exists a direct, as well as a consensual, pupillo-dilating reflex in man; this indicates that a partial decussation of the pupillo-dilation fibers must occur higher up, and it is quite possible that this happens in the dorsal tegmental decussation of Meynert. It appeared logical to look for the crossing of the sympathetic fibers in the commisura cerebri-posterior, which Ramon y Cajal considers an optic reflex path; but we know, through the experiment of Harris,[11] that its cutting is not followed by miosis, and we must therefore conclude that at least the majority of the pupillo-dilating fibers do not take this course.

The question of where the sympathetic fibers have their source is as yet unsolved. Histologic researches of Ramon y Cajal show that the fasciculus bigeminalis connects the lenticular region with the corpora quadrigemina anteriora. Ramsay Hunt [12] came to the conclusion that these fibers of the ansa peduncularis originate exclusively from the globus pallidus, i. e., from those parts of the nucleus lenticularis which, in the realm of motion, plays the same important rôle, through control and regulation of automatic and associated movements, as the thalamus opticus in the domain of sensation, and both of which lost phylogenetically their early importance with the development of the cortex cerebri. Testut tells us that part of the ansa peduncularis enters the fasciculus longitudinalis posterior, which has perhaps for the basal motor ganglions the same importance as the pyramis for the cortical motor region. These pallidal fibers govern the principal movements of Munk, as do the pyramidal fibers with Munk's separate movements. Ramsay Hunt concluded that the fibers originating in the globus pallidus have a purely motoric function: this means that those which run down in the fasciculus longitudinalis posterior dilate the pupil. Consequently, their interruption would be followed by miosis.

Karplus and Kreidl [6] determined by experiments with cats and apes, a center for pupillo-dilation, situated in the hypothalamus, near the dorsomedial portion of the foot of the cerebral peduncle, in the frontal part of the corpus subthalamicum. They could further show that the

11. Harris: Binocular and Stereoscopic Vision in Man and Other Vertebrates with Its Relation to Decussation of the Optic Nerves, the Ocular Movements and the Pupil Light Reflex, Brain **27**:107, 194.

12. Hunt, Ramsay: Progressive Atrophy of the Globus Pallidus, Brain, **40**:58, 1917.

irritation of this center in apes had a stronger effect on the heterolateral sympathetic cord. Their center corresponds, probably, to the corpus of Luys, limited beneath by the pedunculi cerebri, medially by the substantia grisea of the third ventricle, above by the thalamus opticus and laterally by the capsula interna. Ramsay Hunt considers it a derivative of the globus pallidus. All the subthalamic centers undergo, after the histologic search of Ramon y Cajal, a partial crossing; the fibers of the corpus of Luys cross just in front of the red nucleus (commissura de Forel). Ramsay Hunt also came to the conclusion that a degeneration of all the fibers originating in the large cells of the globus pallidus is followed by a syndrome of paralysis agitans, i. e., rigidity, tremor and loss of the automatic function of the corresponding somatic muscles. Another peculiarity of the Argyll Robertson pupil is that it remains not only immobile to light, but also loses the constant and automatic small oscillating movements which the Germans call "Pupillenunruhe"; the amaurotic pupil is also immobile on light incidence but never loses its "Pupillenunruhe," nor becomes miotic. The fact that an Argyll Robertson pupil is not influenced by a retinal process, e. g., that it remains miotic, although the patient becomes blind later by optic atrophy, indicates clearly that the Argyll Robertson pupil is not only produced by interruption of the afferent optico-sensoric path, but by still other etiologic factors. Furthermore, the Argyll Robertson pupil not only grows stiff, but its dilatation becomes difficult by reason of atropin. One can therefore consider the possibility that the peculiar stiffness of the Argyll Robertson pupil is caused by an interruption of striospinal fibers traveling in the fasciculus bigeminalis and their continuance in the fasciculus tecto-spinalis. Against such acceptance, we must say that bulbar as well as spinal miosis are as little followed by rigidity of the pupil as the amaurotic light immobility.

Nevertheless, we must accept the fact that the peculiar stiffness of the Argyll Robertson pupil is produced by a lesion of the striothalamo spinal system, because rigidity plus paralysis are as typical for lesions of the globus pallidus as is spastic paralysis for lesions of the pyramid. Perhaps it is produced by interruption of those fibers which enter in connection with the deepest layers of the corpora quadrigemina-anteriora. We may also remember here that we not seldom find in old people miotic and rigid pupils without loss of light reflex; this is perhaps caused by senile vascular changes in the lenticular region, whereas a lesion in the dorsal tegmental decussation conduces to the Argyll Robertson pupil, namely: to loss of light influence through interruption of the tectobulbar fibers,[1] to miosis through interruption of the tectospinal fibers, to a rigidity through interruption of the

pallidal fibers. If these deductions are correct, we are forced to accept that the crossing of the latter two fibers takes place higher up in Meynert's crossing than the partial decussation of the first named fibers. Also, we will find the following homology between the paths for pupillo-constriction and pupillo-dilation:

Path for Pupillo-Dilation	Path for Pupillo-Constriction
AFFERENT	
Skin corpuscle	Cones and rods } Retina
Spinal ganglion	Bipolar cells } Retina
Nuclei of Goll and Burdach	Ganglion cells } Retina
Sensible crossing in medulla oblongata	Chiasma
	Nervus opticus
Cells of the globus pallidus and corpus de Luys	Corpora quadrigemina anteriora
EFFERENT	
Fasciculus bigeminalis plus fasciculus tecto-spinalis	Fasciculus tecto-bulbaris
CROSSING OF MEYNERT	
Centrum cilio-spinale	Nucleus oculo-motorius
Ganglion, superior cervical	Ganglion ciliare
Dilator iridis	Sphincter iridis

DISSOCIATION OF THE FUNCTION OF THE SEMICIRCULAR CANALS

The patient does not show any signs of disease of the nervii acustici, but there is doubtless a dissociation of the function of the semicircular canal. The examination after the method of Baranyi, with warm water, discloses that the vertical canals do not react, either with nystagmus, vertigo, past-pointing or nausea; whereas the horizontal canals react in every way, though in a lesser degree, as shown by the reduced amplitude and the reduced duration of the experimentally provoked nystagmus. Such a dissociation of the semicircular canal can be caused, according to experience, only by a lesion of the brain stem. We know, through the systematic investigation of Jones,[13] Mills and Weisenburg, that the vertical and the horizontal semicircular canals send their fibers through different paths to the bulbar nuclei, to the cerebellum and to the cortex. The horizontal canal fibers divide before the magnocellular Deiter's nucleus, and send their cerebellar fibers, which correspond to the unconscient muscular fibers of the body, through the inferior cerebellar peduncle; the vertical canal fibers go higher up into the pons (perhaps to the triangular nucleus of Obersteiner) and send their cerebellar fibers through the middle cerebellar peduncle. Our patient lacks from the vertical semicircular canals, not only nystagmus caused by the fibers which go to the ocular nuclei, but also vertigo caused by the fibers which ascend together with the conscient muscular sense and superficial sensibility through the medial laqueus to the cortex,

13. Jones: Equilibrium and Vertigo, Philadelphia, J. B. Lippincott Co., 1919.

and which produce the sensation of vertigo by the difference of the conscient muscular sensation and the unconscient cerebellar muscular-tonus. The patient shows further a lack of normal past-pointing produced by the fibers which go to the cerebellum and cause the unconscient muscular-tonus. It must, therefore, be accepted that the fibers of the vertical semicircular canals are blocked in their totality. It would be quite easy to explain this loss of function if it were found only on one side, because all the other symptoms indicate anatomic lesion in the middle story of the pons just in the neighborhood of the entrance of the nervus vestibularis. Such a unilateral loss has been clinically observed by Jones [13] in a man who, according to the opinion of Weisenburg, suffered from a typical lesion of the middle cerebellar peduncle: the left vestibularis was completely normal; the right one showed only the loss of function of the vertical semicircular canal. The patient in my case showed the loss of function on both sides, and the above deduced anatomic lesion cannot explain directly the loss of the second vestibularis, as we must expect from the cerebellar fibers that they ascend through the homolateral middle cerebellar peduncle. The only manner in which we can explain such bilateral loss is the following: The vertical fibers ascend higher up in the pons and run, therefore, a longer way beneath the floor of the fourth ventricle than the more numerous horizontal fibers; by their longer course they are more exposed to a blocking through an increase of the intraventricular tension. Already Wells P. Eagleton [14] has pointed out that a reduced excitability of the vestibular apparatus of the apparently healthy side is produced by an increase of intracranial pressure. Jones came to the same conclusion as a consequence of numerous careful investigations. Both could observe that a decompressive operation reestablished the function of the vertical semicircular canals. An excellent illustration is one case observed by Jones: [13] An apparently healthy man complained of headache; an examination of the vestibular apparatus revealed loss of the function of the vertical semicircular canals on both sides; irritation of the horizontal canal produced nystagmus but no vertigo; both acustici were normal and repeated neurologic examinations were negative. Some days later the patient died and necropsy disclosed abscess of the fourth ventricle. The exact investigation of the vestibular apparatus dates back only several years, since its introduction by Baranyi; we have, therefore, only a few observations on

13. Jones: Equilibrium and Vertigo, p. 124.

14. Eagleton, Wells P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin, Laryngoscope **23**:592 (May) 1913.

13. Jones: Equilibrium and Vertigo, p. 58.

dissociation of the vestibular function. Jones, however, found thirty-eight among 700 cases on which he made clinical investigations, which perhaps indicates that in the near future numerous similar observations will be made. In all his cases the acustici were normal, the horizontal canals reacted, and the vertical canals showed a partial or complete unilateral or bilateral loss of function. We are not yet able to interpret exactly its bilateral loss. Eagleton, as well as Jones, could also have made the observation that tumors of the cerebellopontile angle block very early, through pressure on the brain stem, but only the function of the vertical semicircular canals of the opposite side is affected.

Therefore, the observation of the eyegrounds with a transition of neuroretinitis albuminuria in a typical choked disk, as well as the bilateral loss of function of the vertical semicircular canals, indicate that the patient in the present case suffered from an increase of intracranial pressure, which probably was produced by an intrapontile tumor causing a crossed hemiplegia and leaving intact the sympathetic pupillo-dilating fibers, because the lesion did not ascend high enough into the substantia reticularis.

I take pleasure in thanking Dr. Fernandez Soto and Prof. José Valdes Anciano for kindly placing literature at my disposal.

ENDOCRINE STIMULATION AS AFFECTING DREAM CONTENT

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REPORT OF A CASE

Following an attack of influenza in January, R. M., a single woman, 45 years old, with a history of previous good health, suffered from lassitude to an extreme degree. She was usually energetic and held a position that necessitated considerable mental and physical activity. Her history up to this time included the usual contagious diseases of childhood, pneumonia, bronchitis and influenza in the epidemic of 1918. The last attack of influenza had been a mild one, with only slight rise of temperature and moderate bronchial and gastro-intestinal symptoms. She remained away from work only two days, and then gradually resumed her usual routine though hampered by great fatigability. About two weeks after this partial recovery, the lassitude still persisting, she applied for treatment.

Miss M. was a stout, well-nourished woman with excellent color and a general appearance of good health.[a] There was no anemia, no sinus involvement or other sequelae to her influenza. She ate and slept well, the cough had subsided, and the gastro-intestinal system was functioning normally. The lassitude was greatest in the morning when she found it extremely difficult to force herself to get up. After she had started to work she felt increasingly better until 4 or 5 o'clock when fatigue began, and she went to bed exhausted about 8. The blood pressure was taken in the early afternoon when the patient was at her best, and was found to be 90, systolic. It was probably much lower at times.

As Miss M's position required long hours of steady application it was necessary to restore her strength quickly, and a course of endocrine therapy was decided on as the best and shortest means of raising the blood pressure and increasing capacity for work. She was given one grain of the extract of whole pituitary gland each morning, beginning the latter part of January. For several days there was no change in her condition but by the end of a week or ten days the blood pressure had risen to 110, systolic, she felt better, began to have a return of initiatve and was able to work her usual number of hours.

Dreams.—About this time Miss M., who had always slept soundly, began to have vivid dreams every night. Previously she had not been conscious of dreaming a dozen times a year, and then only after eating heartily just before retiring. She had always fallen asleep promptly, but after ten days of administration of pituitary extract, as soon as her head touched the pillow she began a series of delightful dream experiences. She began to anticipate going to sleep. The dream adventures were actually commonplace as she remembered them in the morning, and she was at a loss to account for the extreme pleasure they gave her and that she always awoke happy. Miss M. is of an active, cheerful disposition, and the few dreams she had had previous to this experience had usually been pleasant though characterless and hard to recall. During the pituitary period dreams followed each other in rapid succession

all night; she felt herself tumbling from one experience to another; and often they were so vivid as to awaken her—always with a pleasurable sensation.

The patient was fond of traveling and frequently spent her vacations this way. The dreams often took the form of a journey. One many times repeated, but always with varied surroundings and coming as a new experience, was that she was starting on her vacation—at the railroad station—sometimes alone, sometimes accompanied by a sister who has often taken trips with her, occasionally by a party of friends to see her off. But in all these dreams the sun was shining, the friends were laughing and joking; she was happy in anticipation of a pleasant journey. In several dreams the patient noticed that the usually dingy station was clean and bright, that the cars were new and freshly painted, the trainmen wore new uniforms and were smiling. Often it was not a vacation but starting to spend Sunday with her mother in the country, the dreamer intensely happy to be able to go. Less often she seemed to have reached her destination and to be wandering among new scenes and people. The latter dreams were much less distinct and the details not so well remembered.

Miss M. could not recollect having ever dreamed of colors until after she had taken pituitary extract, but in this whole series of dreams colors were bright and distinct; no one predominated over the others. One dream at this time was so vivid that it still seems like an experience of waking life. Miss M. was in the dining car of a railroad train crossing the plains between Chicago and the Rocky Mountains. She was certain of the locality, which she had visited several times. She was looking out of the car window at the flat lands enjoying in anticipation the mountain scenery she expected to reach next day. As in all of these dreams, the colors left the most distinct impression—the bright sunshine, the yellow fields of grain, the deep blue of the sky, the white clouds, the green grass. In the car the silver on the table glittered, the cloth was very white, the mahogany woodwork was almost cherry red. Her companion at the table was of so little account that in the morning she could not remember whether it was a relative or chance acquaintance of the journey. Only one dream in this series was of a different character. In this she was with her married sister, who has children, and her mother. Miss M. dreamt that she was sitting with her mother and sister and that she was expecting the birth of a child. She suffered no pain and the dream was principally occupied by a discussion with the married sister as to whether it were possible to have a baby so easily. The patient has seen women in labor and realized in her dream the unusualness of her experience. There were no details of the birth, but she woke happy and triumphant because she would have the baby with so little trouble. In this dream there was no thought of a father for the child, the patient and her mother and sister taking her condition as a matter of course. Miss M. is very fond of children and has often talked of adopting one.

The principal characteristics of this series of dreams were sensations of light and color—of action, going somewhere—and of pleasure in anticipating happiness to come. They were recalled by the patient as a succession of vivid pictures. Her memory of actual events was always in picture form to a much greater degree than the average. The predominant emotion of the dreams—hope—is characteristic of pituitary activity. The unreasoning depression of hypopituitary patients and their ready response to glandular therapy, their rapid return to a cheerful outlook on life, is in exact accord with the experience of Miss M.

Certain physical symptoms now supervened. By the time she had taken pituitary extract three weeks the patient developed a rather coarse intention tremor of the hands and her thumbs were occasionally sharply adducted. The latter symptom began a few days before the tremor, in the right thumb, later attacking the left one, and was very annoying, making it especially hard to hold a cup without overturning it. She was feeling so much better, however, that she did not report to her physician but continued taking the pituitary gland for a week or two longer. When she did present herself both symptoms were pronounced. The pituitary extract was discontinued, but as the blood pressure was still 110 and as the patient was easily fatigued, one grain of extract of suprarenal (the whole gland) was given each morning and a half grain at noon.

A week after the administration of pituitary extract was discontinued the tremor and adduction of the thumbs became less marked though the former recurred from time to time for several weeks. At about this time the patient suddenly felt well. She went to bed very tired as usual, but awoke in the morning able to get up immediately and begin the day. Until then she had found it impossible even to dress until at least an hour after her breakfast, which at this time included strong coffee. Her blood pressure was taken and found to be systolic, 120; diastolic, 75.

The dreams continued after the administration of pituitary extract was stopped; but a few days after beginning the administration of suprarenal gland they changed their character. They were less vivid and not so easily remembered, there were no colors, and the dreams were without exception unpleasant. Toward the end of the course of suprarenal extract the patient would wake two or three times during the night quite rigid, with a sensation of horror, her muscles so tense that they would ache for some time after waking. Usually the dreams were vague and only the emotions they called forth, fear or anger, would be clearly remembered. Occasionally she dreamt that she was awaiting news that she knew would be terrible. Twice she dreamt that she had violent quarrels. One dream of this character was so real that she awoke shaking with anger against a fellow-worker and could not sleep again for hours. In this dream the whole course of the quarrel, which was a verbal one, was vividly remembered though its cause was forgotten. Once she dreamed that she had committed some crime and was hiding from the police who finally captured her. In this dream she was not in her own identity, though who she was she could not recall, except that from the surroundings and events of the capture she thinks she must have been a man. The crime for which she was apprehended was likewise vague, something to do with a bank.

One of the most distinct dreams of this series occurred after attending a circus and seeing dwarfs. She thought that a horribly misshapen child was clinging round her neck, and that she was unable to loosen its arms. In this dream there was no sense of being strangled. She realized, while struggling fruitlessly to release herself, that it was only the mental horror of having so hideous a creature near her that caused her fear.

After taking the suprarenal gland extract for ten days the patient, who had always menstruated regularly, had a slight flow halfway between her periods lasting two or three days. As by this time she was feeling entirely well and was evidently profoundly stimulated by the endocrines, all medication was stopped. In about a week the dreams ceased altogether, becoming less frightful within two or three days after the suprarenal extract was discontinued. The blood pressure dropped to systolic, 110; diastolic, 70, and

the patient felt a slight return of her old lassitude. The only other symptoms at this time was the appearance of two or three small moles that persisted several months but had atrophied by the time this account was written.

Two weeks after the abnormal menstrual discharge and about a week after the dreams had ceased, the patient had a normal menstruation lasting six days as usual. The dreams recurred and continued through the entire period. They were all pleasant dreams, resembling those that occurred while she was taking pituitary extract. In the one most distinctly remembered she was in her bedroom dressing for some evening entertainment. As usual in these dreams she was very happy and decided to dress her hair elaborately to express this feeling. She arranged it with red and yellow ornaments, bright and glowing, standing out in a sort of halo round the face. She was pleased to see that her hair looked better than ever before. The rest of the family, when she left her room ready to start, exclaimed at the daring headdress and tried to persuade her to change it; but knowing it was becoming she refused, and the dream ended with her starting for her evening's pleasure triumphant in the consciousness that she looked her best.

During the next three months the abnormal menstrual discharge occurred with exact regularity halfway between the normal periods, growing less each month until it was only a stain. It was never accompanied by dreams. Each normal menstruation, however, was marked by a recurrence of agreeable dreams which grew less vivid and fewer each month until they ceased altogether. After the slight regression following the discontinuance of the endocrine administration the general health of the patient steadily improved and for the past two months she has felt perfectly well, with a blood pressure of systolic, 120; diastolic, 80; to systolic, 126; diastolic, 80. Her old capacity for work has returned.

SUMMARY AND COMMENT

In this case we have a woman who has seldom been ill enough to require treatment, aside from the acute infections; she once worked eleven years without losing a day through illness. She is overweight but otherwise in good condition; she has never suffered from any nervous disease. Interest in the case lies in the effect of stimulation of the endocrine glands on the dream content. Aside from the slight depression and apprehension caused by lack of strength in one who must work to live, the patient's mental and emotional condition was as usual. Yet after a purely physical disturbance with overstimulation of the sexual system, as the interference with the menstrual cycle showed, there appeared a series of dreams which, if their cause were unknown, would seem to point to a profound emotional disturbance.

The hypophysis, perhaps our chief stimulant to action through desire, was in this case augmented artificially. During the day there was little appreciable increase in its functions. The patient was overtaxing her strength at the time and noticed only a slight increase in energy while taking it, but at night—when there were no calls on the body beyond its diminished powers—the augmented pituitary gland gave symptoms of overactivity of its normal functions, which included ovarian stimulation. Thus the cause of the dreams was not psychic

trauma, but the result of too great pituitary activity which stimulated the ovaries and raised to the surface of dream consciousness some of the many unfulfilled desires present in us all.

The highly emotional, disagreeable dreams that followed those of frank desire on the administration of suprarenal substance, illustrate the action of this gland as the physical basis of fear and its kindred emotions, and these dreams are capable of the same explanation as the wish dreams. It is interesting to note that Miss M. was not emotionally affected by the endocrines during her waking hours and actually felt more cheerful while under the influence of suprarenal substance because, as her blood pressure increased, she was better able to work and ceased to worry about her health.

As living creatures increase in complexity and specialized organs appear the gonad system develops as the physiologic means through which the organism reacts to emotions. Pathologic overactivity of the various endocrine glands is capable of autonomously calling forth emotions without, or wholly disproportioned to, any foreign stimulus. In the case of Miss M., two of the glands controlling desire and fear were artificially augmented and the ensuing condition was the same as that produced when one or the other of these glands is over active per se. The picture is clearer than in most cases because the augmentation of one gland has not been caused, as it so often is, by the hypoactivity of another. This case illustrates through the dream the physiologic control over desire and fear, the two great factors in human conduct.[1] It is a good example of the purely physical origin of many dreams and the emotions behind them; an aspect of the subject that has been somewhat lost sight of in the fervor of Freudian interpretation.

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1. Bailey: Desire and Fear as Factors in Human Conduct, President's Address, Transactions of the American Neurological Association, 1912.

News and Comment

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The first annual meeting of the Association for Research in Nervous and Mental Diseases, held in New York City, Dec. 28 and 29, 1920, was very successful. The subject of lethargic encephalitis in its various aspects was discussed in about thirty papers. These articles, together with questions by members of the Commission on Scientific Work and others and the answers by the readers of the papers, will be edited and published by the commission.

The officers elected for the ensuing year are: president, Walter Timme, New York; vice president, E. W. Taylor, Boston; secretary-treasurer, Foster Kennedy, New York. Members of the Commission on Scientific Work, in addition to the officers, are: William G. Spiller, Philadelphia; Hugh T. Patrick, Chicago; Charles L. Dana, Bernard Sachs, Israel Strauss and J. Ramsay Hunt, New York.

The next meeting will be held in New York City during the Christmas holidays of 1921.

Abstracts from Current Literature

SOME MORPHOLOGIC DIFFERENCES IN SPIROCHAETA PALLIDA IN THE BRAINS OF GENERAL PARALYTICS, WITH ONE TABLE OF ILLUSTRATIONS. George Springer, Arch. f. Psychiat. **61**:479, 1920.

The author summarizes the observations of earlier writers on the length and size of the spirochetes and on the length and thickness and mode of curving or bending of their spirals. He points out the necessity of more constant and methodic study of these conditions in order to establish some general laws in regard to the varieties and diversities of spirochetes found in the human brain in general paralysis of the insane.

Springer reports a case of typical paralysis which, however, from the early age of the patient at its onset might possibly fall into the class of juvenile paresis. In this case the spirochetes were present in great numbers, and the greater part were unusually long and slender, being from two to three times the length of spirochetes ordinarily seen in brains in general paralysis. The spirals, which generally show from 8 to 13 turns and occasionally as high as 20 to 24, in this case attained from 26 to 30 and even as high as 32 turns. The turn of the spiral was generally somewhat flattened not only in the middle of the organism, as commonly occurs, but throughout the whole length. While Noguchi gave 0.2 m. as the thickness of the thinner examples seen by him in contrast with his two other varieties at 0.25 and 0.3 m. in thickness, Springer's thin variety shows only 0.15 m. at the most. The spirochetes were so fine and so long that they gave the impression of a tangle of the finest of threads on a carpet. In addition to their extreme tenuity they underwent sharp bends and irregular windings. These appearances were not caused by shrinking of the tissues or other accidents of the tissues of the preparation since side by side with the sharply bent and irregular organisms others were seen of perfectly regular outline. Although flattened spirals were the rule in this case, small numbers of organisms were found scattered among those described which resembled the normal picture of the spirochete, with broad and thick turns of the spirals. But even in these forms the spirals were from 26 to 30 in number. They were found, as a rule, in thick groups or swarms of other spirochetes, and in the same localities short and thick forms were seen in small numbers and small round dark-staining bodies were also seen often attached to a spirochete. These small objects were from one to four times the diameter of a medium sized spirochete, and the author is certain they were not in any sense artefacts. In general, they were in contact with the shorter and thicker spirochetes and not with the long, thin variety. In thick clumps of spirochetes many were seen of normal length and size; in fact, the thicker the clump, the nearer normal in length, thickness and type of spirals were the spirochetes. The small balls may be permanent forms of the organism.

In some swarms of spirochetes large numbers were seen which looked extremely fragile, mostly fine pointed rod forms lying in a row; in their neighborhood were seen short, but still spiral forms which were made up of many little block fragments of varying size. Short spirochetes were also seen of normal form and medium size in thickness, but with very few turns of the spiral. The small fragments, however, would appear preeminently in such a

picture. In serial sections such a clump would show throughout a whole block, showing that they were by no means an accidental product. In such a swarm, many of the little balls spoken of were seen. Sometimes many individuals in such a clump did not stain normally, but were yellowish and brownish.

Rolled up forms were seen in moderate numbers and in general limited to the larger masses of spirochetes. They were rolled at one or at both ends; buds, ring and star forms were also seen. Whether there is any connection between the small round bodies and the buds, and whether the thick rings represent the union of two individuals which frequently has been seen in the form of a twisted loop is difficult to determine.

The medullary portions of the brain were free from the spirochetes. All layers of the cortex contained them, but the fine threadlike forms were most common in the more superficial layers. The greater number of swarms and clumps were found in the outer layers, but the fifth and sixth layers also showed a certain number of these conglomerations. Frequently considerable areas would show diffuse localization of the spirochetes only and frequently in small numbers. The parasites often lay in little coils of long threads, sometimes surrounding glia cells and more rarely, ganglion cells. With the larger masses, of which from one to two dozen would lie in a microscopic field which showed a practically diffuse localization, more rarely thick sheaths around the capillaries were seen, as described by Jahnel.

The author claims no priority for the foregoing descriptions, but considers that every well studied case should be presented in detail for the purposes of study and comparison by the observers.

In resuming, the author says: "The biological alterations in protozoa of which the change of form is one of the most easily recognized are dependent on internal and external factors. It is to be supposed that certain changes in the exterior media, as, for example, the artificial culture media and also the products of syphilitic disease, may bring about marked changes in the form of the spirochaeta pallida which, indeed, shows that the body has a remarkable power of change, but does not justify the assumption that it has the power of forming a particular sort of an independent variety."

To prove whether certain varieties are transient in character or are independent and capable of being inherited, experimental methods of propagation are of more value than the observation of morphologic characteristics. Investigations in this line seem to show that all effort to change a "pure" race so that the new characteristics are hereditary are unavailing; that although great variations are seen a certain normal "mean" is retained. The author's observations on the spirochete tend toward the same result as regards the spirochete in syphilis, and he concludes that marked variability of the *Spirochaeta pallida* exists not only from a morphologic, but also from a biologic standpoint. In all probability, the spirochete is incapable of transmitting the new characteristics to descendants.

GURD, Ann Arbor, Mich.

A POLYNEURITIC SYNDROME RESEMBLING PELLAGRA-ACRODYNIA (?) SEEN IN VERY YOUNG CHILDREN. ALBERT H. BYFIELD, Am. J. Dis. Child. **20**:5 (Nov.) 1920.

This is a report of seventeen cases of an epidemic erythema in children all of whom are under 4 years of age. The studies extend over a period of five years.

Clinically, these patients exhibited many interesting symptoms and signs. Predominant were the trophic changes found in all. There was alopecia, falling out of the teeth, reddening of the skin, neurokeratitis and changes in the nails. In the nervous system there were paresthesias of the hands and feet, diminished cutaneous sensibility in some, constant rubbing of the skin, pulling out of the hair, reflexes diminished or absent, muscular weakness and atrophy. Mentally the patients exhibited dulness and apathy alternating with restlessness and sleeplessness. In bed they assumed a fairly characteristic posture, lying on the side with the head buried in the pillows. They presented a picture of extreme wretchedness. The skin evidenced vasomotor disturbances in cyanosis and lowered temperature in the extremities. There was a nonconfluent erythematous rash recurring at intervals of about two weeks, which somewhat resembled pellagra. Desquamation was marked on the tips of the fingers and toes, and in some cases it was also seen on the thorax and upper arms where the lesion presented a morbiliform or bullous appearance. In the digestive tract, there was obstinate anorexia, but no vomiting or diarrhea; there was also some constipation. In the advanced cases there was unusual sweating. In the genito-urinary system, there was urinary frequency and often pyelitis. The laboratory examinations showed a consistent leukocytosis of 11,000 to 30,000 but no predominance of polymorphonuclears in the differential count. The stomach contents were invariably negative. The urine showed acetone, and, during the pyelitis, pus cells. The spinal fluids were negative with the exception of increased globulin. The Wassermann tests were negative.

The duration of the disease varied from two to eight months, and fourteen of the seventeen patients recovered.

A careful survey of the family histories and other facts failed to reveal any dietary deficiency; in fact, some of the patients were breast-fed at the time of the onset, and the mothers' diets were adequate. Many cases began as bad colds or influenza. Diphtheria-like organisms were found in many throat cultures but the patients gave a negative reaction to the Schick test, and the course of the disease was not altered by the administration of diphtheria antitoxin. No hereditary influences could be found. Seasonal distribution was the nine months between October and July. The fact that all presented leukocytosis argued for an infection—in fact, two cases cleared up after tonsillectomy and one after a nasal surgical operation.

The diagnostic possibilities were numerous. Pellagra was considered, but there was no sharp line of demarcation in the skin lesion; no common dietary error; no history of pellagra in the family; the age of the patients was uniform; there was insufficient digestive disturbance; lack of recurrences; frequent history of infection; occurrence in breast-fed babies; aggravation of symptoms in cold weather and prolonged duration of symptoms. Motor neuritis was ruled out because of the preponderance of sensory phenomena. Arsenic poisoning and ergotism were easily excluded. Trophoneuroses offered slight fundamental similarity, but differed as to age incidence. Acrodynia or "epidemic erythema" (Weston) is proposed, because of its close resemblance, but all reports of this disease show that the patients had conjunctivitis, spasticity and convulsions. The possibility of a postinfluenzal condition, such as a radiculitis or an infectious polyneuritis, is considered and supported by bibliographic reviews, but left merely as a consideration.

As for the treatment, gavage and forced feeding were found to produce the best results, and, under this routine, improvement began and continued favor-

ably in fourteen cases. Forced feeding was not attempted in the three fatal cases as they were early cases and the patients died in a short while. One patient died of pulmonary complications.

PATTEN, Philadelphia.

ERGEBNISSE DER BLUT UND SPINALFLUESSIGKEITS-UNTERSUCHUNG BEI LUETISCHEN HIRN UND RUECKENMARKS-ERKRANKUNGEN (RESULTS OF A STUDY OF THE BLOOD AND SPINAL FLUID IN SYPHILITIC DISEASE OF THE BRAIN AND SPINAL CORD). HERMAN KRUEGER, Monatschr. f. Neurol. u. Psych. **48**:18 (July) 1920.

The value of the "four reactions" of Nonne—cell count, globulin, spinal fluid Wassermann and blood Wassermann—is beyond question. Nevertheless, practically all series of studies have shown that there are cases of central nervous system syphilis, proved clinically, therapeutically and pathologically, in which some or all four reactions are negative. As Rost has pointed out, negative reactions do not exclude syphilis; parenchymatous, endarteritic and even meningeal foci may exist without demonstrable fluid change. Even in general paresis, in which we are most likely to find all reactions positive, negative reactions may occur. These cases are sufficiently numerous to detract greatly from the diagnostic significance of the four reactions.

If all four reactions are positive, we are dealing with syphilis. It may involve the substance of the brain and cord, or it may be confined to the meninges (as is probably true in many of the secondary cases with positive fluids).

The author reports the results of the study of the four reactions in 310 cases of general paresis and taboparesis, eighty-four cases of tabes dorsalis and seventy-two cases of cerebrospinal syphilis.

General Paresis and Taboparesis.—The blood Wassermann reaction was positive in 96 per cent. of the cases; the spinal fluid Wassermann reaction in 93 per cent.; pleocytosis (over 10 cells) was present in 97 per cent., and globulin in 99 per cent. All reactions were positive in 88 per cent. of the cases. In no case were all reactions negative. In 12 per cent. of the cases, one or more reactions were negative. These negative reactions were not necessarily found in incipient cases, stationary cases, or cases in remissions. Many were typical cases, running a typical course, some of them rapidly progressive. In no case was it possible to render all the reactions negative by treatment.

Tabes Dorsalis.—The blood Wassermann reaction was positive in 85 per cent. of the cases; the spinal fluid Wassermann reaction in 77 per cent.; pleocytosis was present in 88 per cent.; globulin in 90 per cent. In 3.5 per cent. of the cases all reactions were negative. The findings were less constant than in paresis, and the effects of treatment more marked.

Cerebrospinal Syphilis (Meningitic, Gummatous and Vascular Forms).—The blood Wassermann reaction was positive in 79 per cent. of the cases; the spinal fluid Wassermann reaction in 66 per cent.; pleocytosis was present in 80 per cent., and globulin in 80 per cent. All reactions were positive in 51 per cent. All reactions were negative in 4 per cent. The effect of treatment on the reactions was greater in this group than in the other groups, and this may be responsible for some of the negative cases. It is not responsible for all of them.

Summary.—1. Negative findings do not exclude syphilis of the central nervous system.

2. With all four reactions positive, the disease with which we are dealing is in all likelihood syphilitic. The Wassermann reaction in the spinal fluid is specific, and if it is positive, it has the same significance as the four reactions in toto. Pleocytosis and a positive globulin reaction are not specific, and occur in numerous other diseases. In case of doubt, the effect of a course of treatment on the spinal fluid will decide.

3. The four reactions in themselves give little help in the differential diagnosis between the types of central nervous system syphilis. We can get some information by watching the results of treatment. Marked improvement in the fluid under treatment is strongly suggestive of cerebrospinal syphilis as differentiated from paresis and tabes.

4. Laboratory findings are valuable, but cannot replace clinical study. One cannot diagnose diseases of the brain and cord in the laboratory.

SELLING, Portland, Ore.

THE PATHOLOGY OF "SO-CALLED" LETHARGIC ENCEPHALITIS.

FELIX STERN, Arch. f. Psychiat. **61**:621, 1920.

In a closely printed article of sixty-seven pages, the author first discusses the proper meaning of the term encephalitis and incidentally that of inflammation, and endeavors to limit the name to certain cases and classes of cases. He then discusses in great detail the histopathology of the four cases that he had examined, going superficially into the clinical histories. He attempts to classify his cases according to the findings in different stages of the disease and in typical and atypical cases, and finally he endeavors to establish a pathologic differential diagnostic syndrome of lethargic encephalitis or, as he prefers to call it, polio-encephalitis (Economo's type). He summarizes his own conclusions as to the histopathology in typical cases somewhat as follows: The picture is that of disease extending over large regions of the brain, but intensified by foci of interadventitial or peri-adventitial infiltration by lymphocytes and their derivatives. plasma cells and polyblasts, and accompanied by alterations in the ganglion cells, acute swelling and advanced degeneration, slighter alteration in the nerve fibrils and marked focal degeneration of the myelin sheaths, with active proliferative changes in the cellular glia and in later stages of the fibrous glia also, and with proliferation of the cellular elements of the vessel walls. The passage of hematogenous elements into the ectodermic tissue is found to a slight degree and some formation of new vessels is seen. Diffuse inflammation of the meninges always takes place, but there is no direct relation or subordination of the encephalitic process to the meningitic. In the brain the gray matter is the area of predilection, especially the thalamus and the central gray matter from the third to the fourth ventricle. Diffuse alterations in the greater part of the nervous tissue of the cortex with glia reactions, without any trace or with small sclerotic foci with reparatory proliferation of the glia and myelin sheath and sometimes slight diffuse gliosis in the area of earlier exudations, are characteristic of the disease. These are the typical accompaniments of the disease. The author looks on hemorrhages, which are usually slight, thrombosis, slight thrombotic softenings and infiltration by polymorphonuclear leukocytes as accidental accompaniments, such as may be seen in many other conditions, and which are fre-

quently absent in encephalitis. Economo's opinion that the leukocytic infiltrations may be rapidly passing initial phenomena is probably correct, and the author calls attention to the fact that in epidemic meningitis also, the primary leukocytic foci are followed rapidly by plasma celled and lymphocytic infiltration. The free gliogenous or angiotic granular cells containing fat are more characteristic by their absence than by their presence in acute cases of lethargic encephalitis except in small thrombotic softenings. Although veins are more constant seats of infiltration than arteries, some arteries are infiltrated, and the predilection of the infiltration for veins is not characteristic of lethargic encephalitis alone as in epidemic cerebrospinal meningitis the veins are the preferred seat of infiltration in the pia and arachnoid.

The cells forming the infiltrative exudate are distinguished with accuracy as lymphocytes, plasma cells, polyblasts (cells with certain resemblances to plasma cells but which do not show the metachromatic staining and the clear "court" of the former cell; they are usually round or ovoid and the nucleus is much darker staining than that of the fibroblasts) and a cell with a round pale nucleus, originating in the vessel walls. The last cell is commonly called epithelioid, but the author objects strongly to the name and hopes it may shortly disappear as it is used indiscriminately for gliogenous elements for Maximov's polyblasts and for the large cells seen in tubercles. A negative characteristic of some value in the diagnosis of lethargic encephalitis is the small number or complete absence of granular (fat) cells, with the exception of those directly attached to the vessel walls.

The author's consideration of the etiology is based on a small number of observations made by his own countrymen, and he ignores the large amount of experimental and clinical observations made in other countries. He brings out rather clearly, however, the fact that there is more than one variety of encephalitis seen in epidemics of influenza.

He explains the predilection of the infiltrative process for the central gray matter of the pons, peduncles, etc., to the terminal character of the arteries of these areas, also to the increase of blood pressure in them caused by the arterial branches coming directly from the carotids, assuming as a primary fact that the blood vessels are the seat of the beginning of the toxic effects of the originator of the disease.

His atypical case was one in which there was a large red softening in the cortex caused by thrombosis of the pial veins, purulent meningitis, multiple hemorrhages and other conditions. Space is lacking for a detailed account of the case of the patient with an abscess in the right lung and pneumonic foci in the left lung, with many bronchiectases and emboli in one branch of the right pulmonary artery. On this case he attempts to base conclusions as to the origin of lethargic encephalitis as the case resembles one described in Economo's report and an organism was found, a "diplostreptococcus" similar to that found by Wiesner in Economo's case. He tries to explain the presence of a leukocytic infiltration and other conditions by the varying action of one and the same organism at different times under different conditions, giving as an example the action of *Spirochaeta pallida* in different forms of syphilis. The most valuable part of the article lies in the attempt to limit the term encephalitis to inflammatory processes and to the careful and detailed description of the histopathology of the four cases given.

The illustrations are not of the usual high standard to which we have been accustomed in German work.

GURD, Ann Arbor, Mich.

SYNDROME OCULO-SYMPATHIQUE DE CLAUDE BERNARD-HORNER PAR COMMOTION D'OBUS — PHENOMENE DE L'ADDUCTION OCULAIRE PROVOQUEE PAR TOUTE EXCITATION PERIPHERIQUE (CLAUDE BERNARD-HORNER OCULOSYMPATHETIC SYNDROME AS A RESULT OF DETONATION SHOCK, WITH OCULAR ADDUCTION ON PERIPHERAL STIMULATION). A. LÉRI and J. THIERS, Rev. neurol. **26**:808 (Nov.) 1919.

In the case reported by the authors, there was a history of loss of consciousness, following shell explosion, on two occasions (July, 1916, and December, 1917) unaccompanied, on either occasion, by evidence of external injury. Following the first accident, persisting auditory defect, on the left, with perforation of the left tympanum, tinnitus, vertigo and headache was noted. Examination, subsequent to the second accident, revealed pronounced vertigo with uncertain, hesitating, lateropulsive gait and very pronounced Romberg symptoms. The left ear showed slight perforation of the tympanum with greatly diminished auditory acuity. The Valsalva sign was positive and the Weber test was localized to the left. The tendon reflexes were present and equal, and there was no clonus or Babinski sign. Sensation was undisturbed, and there was no sign of disorder of cerebellar function. The left pupil, however, was much smaller than the right although neither showed disturbance in reaction to light or accommodation. There was slight exophthalmos on the left, and the palpebral fissure on this side was narrower than on the right — in effect, the Claude Bernard-Horner oculopupillary syndrome. It is of interest to note, in this connection, that Cobb and Scarlett[1] report *enophthalmos* in each of the eleven cases of Claude Bernard-Horner syndrome comprising their series. It might be added that in Léri and Thiers' case, vision was apparently normal in both eyes and no disturbance was noted in ocular movement save that there seemed to be slight instability of the eyeballs in direct vision, and occasional nystagmoid movements were noted on the direction of the gaze laterally upward and to the right. The Wassermann reaction was negative, and there was no history or clinical evidence of syphilis.

On later examination, in connection with the Bárány test, it was noted that when cold was applied to either external auditory canal, there appeared, uniformly, a very definite syndrome characterized, objectively, by complete adduction of the left eyeball, contraction of the superficial muscles of the right cheek, and, finally, failure of equilibrium and falling. Subjectively, there were noted intense vertigo and diplopia. On further investigation, it was found that this syndrome could be produced by practically any type of peripheral stimulus, thus application of cold to the nostril or to the buccal mucosa, pinching of the skin, electrical stimulation, etc. In attempting an explanation of the mechanism underlying the production of this reaction, the authors direct attention to the possible causative relation of the existing labyrinthine defect, occurring as a result of the detonation-shock, citing, in support, a case in which there was observed a similar reaction, although much milder in type, and in which there was a definite history of labyrinth injury as a result of a fall from a horse.

RAPHAEL, Kalamazoo, Mich.

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1. Cobb and Scarlett: Arch. Neurol. & Psychiat. **3**:636 (June) 1920.

SOME RECENT RESULTS IN THE INVESTIGATION OF THE RELATIONS OF SPIROCHETES IN THE BRAIN IN GENERAL PARALYSIS OF THE INSANE. F. JAHNEL, Allg. Ztschr. f. Psychiat. **75**:503 (May) 1919.

The author reviews the work already done in investigation of the spirochetes' presence, distribution, etc., in general paralysis of the insane, and calls attention to the sources of failure always to find the organism, namely, their presence in swarms like bees in certain areas only, their selection of certain areas in a given case to the exclusion of other areas, and their periodic increase and decrease in number. The last cause is the expression of a biologic characteristic of all spirochetes that are active in inducing illness. The spirochetes do not entirely disappear at any given time, but become so reduced in number that great difficulty is experienced in isolating them. The author has established this phenomenon not only by clinical and microscopic observation, but also by experimenting with large amounts of blood injected at the time of the lessened number of spirochetes, and he concludes that spirochetes are present at all times in all cases of general paralysis of the insane although he may be able to show their presence in a part only of all cases. The author's present efforts are not toward obtaining spirochetes in all cases, but to determine the localization of the spirochetes in a large number of cases and the relations of the spirochetes to the tissues and the conditions under which the organisms arise and multiply in the nervous system.

The author's earlier works have identified two types of localization of the spirochete, in foci or disseminated. The present work adds a third localization, that of the vascular type in which the cortical vessels are specially the seat of the organisms. The spirochetes are not found in relation to all the vessels in a case, but usually all the vessels in a circumscribed area show the invasion of their walls by the organisms. Both veins and arteries show sheaths of the germs which also penetrate the vessel walls and lie in thick masses, sometimes lengthwise at other times crosswise. The parasites are frequently in such numbers that under a low power the vessels invaded by them appear darker than other vessels, and in oil immersions the vessels appear black, and it is only on using the fine micrometer that the individual spirochetes come to view. The cortical parenchyma is not entirely free from the spirochetes in these cases, but their number is extremely restricted and varies directly as the distance from the vessel wall increases. Sometimes the perivascular sheath is so excessive that the parasites invade the surrounding tissue and by the union of several of these small foci one of the large or "swarm of bees" type of focus results. On the edge of one of these giant foci the vascular type is frequently seen in its purest form.

The capillaries are invaded by the spirochetes in the same manner as the larger vessels. It is impossible to ascertain the relations of the spirochetes to the different vessel walls as the great number of the organisms prohibits close observation. Sometimes the spirochetes are in such numbers that the vessel appears like a tube composed entirely of spirochetes. Sometimes one portion of the spirochetes lies in the vessel lumen, at other times one portion is affixed to the vessel wall and the other portion lies in the parenchyma. At branching of the vessels the spirochetes frequently form a wedge or funnel passing from one vessel to the other. When marked infiltration of the larger veins by plasma cells and lymphocytes is seen, the spirochetes are seen between

these cells. The spirochetes pass through all the walls, even the intima is so invaded by them that it stands out as a blackish wall formed by concentric rings of spirochetes.

The vascular type shows certain interesting characteristics. Sometimes the spirochete sheath around a vessel ceases abruptly, sometimes gradually; at some divisions of the vessels many spirochetes are seen, at others few or none; there sems to be no regularity in these relations. Large veins often show a spirochete sheath passing only half or three-quarters way around its lumen, and the other half or quarter will show only scattered organisms.

An important point seems to be that when the vascular type is found well marked, it is seen in all portions of the brain and not only in a limited area. The white substance seems to be exempt from the spirochete as a rule, but in one case the author found spirochetes in the perivascular spaces in the white substance. The organisms are frequently found directly under the pia, in or under the glial limiting membrane. As a rule, the molecular layer is free from parasites, but sometimes when they are plentiful in the next layer a few wander into the molecular layer.

In one case, foci were found directly under the pia and in the same case "bee swarm" foci and also disseminated spirochetes were found in the ganglion cell layers. In one case spirochetes were seen forcing their way into the pia. This is probably a rare occurrence.

Two clinical histories are related in detail with resultant necropsies, both showing the vascular type of spirochete localization, but the clinical courses of the two cases and the degree of invasion and degeneration of the parenchyma were so utterly dissimilar in the two cases that no possible conclusions could be drawn either as to symptomatology or as to the severity of the process in cases of vascular localization.

The question arises whether the vascular type of localization may not be an intermediate term of a later "bee swarm" foci arrangement, and one is led again to a consideration of the path by which the spirochete enters the brain. Spirochetes have been seen in the blood, but may it not be that the lymph stream alone carries them to the brain, which the marked vascular localization would render probable, and their escape again into the blood stream would explain their presence there? All these interesting details and questions presented by the author must incite to a more general and thorough study of the whole question into which light can only be brought by large numbers of detailed observations. The author's figures are extremely conservative in regard to the percentage of cases in which he has found spirochetes. Taking a series of unselected cases, he found spirochetes in the dark field in 50 per cent. and in cut sections in only 25 per cent.

GURD, Ann Arbor, Mich.

DU TRAITEMENT DE LA PARALYSIE GENERALE PAR L'INOCULATION DE CERTAINES MALADIES FEBRILES (PALUDISME —FIEVRE RECURRENTE) (TREATMENT OF GENERAL PARALYSIS BY INOCULATION OF CERTAIN FEVERS — MALARIA, RELAPSING FEVER). P. PAGNIEZ, Presse méd. **28**:736 (Oct. 16) 1920.

This paper is chiefly a review of German work published since 1918, particularly that of Mühlens, Weygandt and Kirschbaum, "Die Behandlung der Paralyse mit Malaria und Rekurrenzfieber," *München. med. Wchnschr.* **67**: 831 (July 16) 1920. The results obtained by these inoculations were not spec-

tacular, but seemed sufficiently encouraging to warrant study and further experimentation.

Among the different fever-provoking agencies tried, tertian plasmodia came to be preferred. From six to eight paroxysms (sixteen in one case) were induced in a course before it was terminated by quinin medication. The maximum body temperature reached was 106.8 F., compared with a maximum of 105.4 and an average of 104 degrees for relapsing fever. The mechanism of the curative process was not clear. Some experiments on animals suggested that the spirocheticidal action of high temperature might alone be responsible for whatever improvement was effected.

Of 33 patients treated, 3 died of intercurrent diseases, 1 died of a complication dependent on the therapy, 12 had treatment discontinued for various reasons, 5 were not favorably affected, while 12 were benefited to such an extent that the improved status could fairly be termed a remission. Some of these paretic patients recovered sufficiently to return to responsible positions, and there carry on their former occupations satisfactorily. Most of these remissions had lasted for several months, and in one case for as long as three years. They exceeded the usual expectation of remissions under other forms of treatment. The serology was variously influenced. Generally blood Wassermann reactions were unchanged, while spinal fluid reactions showed more or less improvement.

In selecting patients suitable for this treatment, it was necessary to exclude all old weakened persons, all cases of long standing, and patients whose quinin tolerance did not show a sufficient margin of safety for the after-treatment.

HUDDLESON, New York.

THE THERAPY OF GENERAL PARALYSIS. R. WEICHBRODT, Arch. f. Psychiat. **61**:132, 1920.

This article contains a critical review of the treatment of general paralysis. The author includes in detail the results of his own researches with therapeutic measures. His conclusions, stated in the introductory paragraph, are that "The therapy of general paralysis is as yet an unsolved problem; the progress of the disease continues and leads with few exceptions to death in from three to five years." With due regard for the frequent occurrences of remissions and the rare cases in which remissions have lasted for years, he comments "theoretically one may rightly believe that such improvements may become permanent and as Spielmayer has stated, from the pathological-anatomical viewpoint there is nothing that excludes the possibility of recovery. Of course, it could only be recovery with defect, but the observations of remissions even in advanced stages show that the brain even in diffuse processes has great possibility of compensation." Remissions in general paralysis stand in close relation to the biologic characteristics of the spirochetes. He refers to the work of Jahnel, which seems to show that as the paralytic attacks are explainable by flare-ups in the increase of spirochetes which after the attack die rapidly, so the remissions may be due to periods in which most of the spirochetes have died, and the few that remain are in an inactive phase. One may only expect therapeutic success by reaching the spirochetes, and only when their increase can be prevented can there be a remission or can the disease be brought to a standstill.

The author presents a chronologic and systematic consideration of the therapeutic efforts that have been reported in the literature. His discussion

of the progress of arsphenamin therapy covers fully the more important contributions dealing with methods and their application. From the experience of the clinic at Frankfort he concludes that there have been no serious bad effects from its use. Now and then there have been improvements, but it is questionable whether these can rightly be ascribed to the therapy. Following a review of the therapeutic effects of mercury, iodin preparations and collargol, he gives in detail some personal researches with methylene-blue and trypan-blue.

He believes that when one summarizes the results of experiences with pharmacologic agents, it is evident that no one of these is able to produce a permanent improvement in the disease. The remainder of his contribution is devoted to a discussion of the therapeutic influence of febrile temperatures on the course of mental disorders and specifically on general paralysis. A review of the extensive literature dealing with this subject forces the conclusion that febrile disease does not always influence a psychosis, but the undoubted improvements and long remissions that have been observed justify a further study of the influence of temperature on general paralysis. He records his own researches in this direction in much detail. He produced febrile temperature with inoculations of the blood of the tertian type of malaria in two cases of well advanced general paralysis. Both patients developed malaria with high temperature, but the course of the general paralysis was uninfluenced. Two other patients in whom paralysis were less advanced improved so much that they were able to leave the hospital; they said that they felt well and that they noticed an improvement of their memory. Too much weight should not be placed on these results as the experiments are recent, but they justify further investigations with this method. As to whether the influence excited by febrile conditions comes from the high temperature or from the hyperleukocytosis, a review of the literature and his own experiments indicate that it is not the result of the increase of leukocytes. From observations showing that intercurrent infectious diseases have a favorable influence on an existing syphilis, the author, working with Jahnel, was able to produce death of the spirochetes in rabbits with syphilitic chancre by exposing the animals to a temperature of from 42 to 43 C. at three intervals of time. This suggested a line of experiments to produce high temperatures in the body of patients with general paralysis. The only way of producing sufficiently high temperature in the body of man is the hot bath and the question arises whether by this means an influence could be exerted to check the growth of spirochetes in the brain. This method of attacking the problem seems hopeful enough to warrant further experiments.

BARRETT, Ann Arbor, Mich.

XANTHOCHROMIA WITH REPORT OF THREE CASES. LOUIS A. LEVISON, Arch. Int. Med. **26**:4 (Oct. 15) 1920.

Xanthochromia is not an uncommon finding in spinal fluids, but a true Froin's syndrome is much less frequent. Yellow fluids occur in many conditions, including general infections, icteroid states and obstructive lesions of the central nervous system. The color is not in itself pathologic, but the coagulability of the albumin content is and does not occur except in lesions in which the circulation of spinal fluid is cut off. Froin's theory as to the color is that it is obtained from bilirubin, but others do not sustain this theory and suggest that it originates from hemorrhage, although no explanation is offered for the

absence of albumin. The important findings then, in a yellow spinal fluid are, increase in the number of cells and the tendency to coagulation. Yellow fluids, however, occur sometimes when no cause can be found.

The author reports three cases of yellow fluids bearing out the preceding discussion. The first case is that of an inoperable intramedullary spinal tumor. The spinal fluid was a rich canary yellow in color; it had 22 cells per c.mm., 18 of which were lymphocytes; an excessive amount of globulin, with partial spontaneous clotting; and negative Wassermann and Fehling's reactions. The second case was diagnosed as syphilitic pachymeningitis with gummatous formations. The spinal fluid was a bright canary yellow, it contained a large amount of globulin which did not coagulate, 78 to 140 lymphocytes per c.mm. and a strongly positive Wassermann reaction. The fluid became clear after a few months of active antisyphilitic treatment. The third case was an example of a yellow fluid in a man with biliary cirrhosis. The spinal fluid was under tension; it was a dull brownish yellow color and showed an increase in globulin but no coagulability; the cell count was 18 cells per c.mm., all small lymphocytes, and the Wassermann and Fehling's reactions were negative.

The author concludes that Froin's syndrome is diagnostic of an organic spinal lesion of the obstructive type, but a mere color change is not important.

PATTEN, Philadelphia.

PSYCHIC DISORDERS IN TABES. KASIMIR BRODNIEWICZ, Allg. Ztschr. f. Psychiat. **75**:701 (Aug. 14) 1919.

While tabes and paresis are commonly associated, there are many cases of tabes accompanied by psychic disturbances that differ from those of paresis in their character and the course of the disorder. The author reviews the literature dealing with psychoses in tabes in which various opinions are expressed as to the development and course of tabes psychoses. It has been held by some that in a reflex way the various neurologic disturbances of tabes may, by influencing the brain centers, bring about the occurrence of a psychosis. Others have asserted that tabes acts only as a predisposing factor for the development of elementary psychic symptoms which may be further elaborated into a psychosis. Anatomic studies have definitely shown that many tabes psychoses are to be considered as part of a general syphilitic or metasyphilitic disorder, which may be an arrested paresis or a mild cerebral syphilis.

The author reports three cases that he had observed in the Rostock Psychiatric Clinic. The first of these was that of a man, 46 years old, who had a syphilitic infection at the age of 19. Tabetic symptoms appeared ten years later. When admitted to the clinic, his pupils were myotic and stiff. The knee and Achilles' reflexes were absent. There were Romberg's sign, marked hypotonia and sensibility disturbances. The Wassermann reaction of the blood was positive. Psychotic symptoms appeared soon after his infection. For a considerable time these were little more than a marked irritability and defective judgment. There later developed a paranoid attitude toward his wife with delusions of suspicions. This subsequently was extended to include others who came in contact with him, and finally led to delusions of persecution which he continued to hold without deterioration of memory or judgment for thirteen years. The second case was that of a man aged 32, who had slowly coursing tabetic symptoms; stiff pupils, disturbances of sensibility and gastric crises. Somewhat atypical were the presence of a Babinski reflex on the left side and

a negative Wassermann reaction of the blood and spinal fluid. There was, however, a slight pleocytosis. At the age of 40 a mild depression developed with an attempt at suicide. In the further progress of the disorder there were delusions of poisoning, states of excitement and delusions of jealousy. His interest in his work fell off, and when released, he was unable to support himself. The third case was that of a man, 52 years of age, who on admission was in the phase of development or remission of a mild tabes. His pupils were narrow and had a slowed light reaction. There was a slight weakness of one facial nerve. The knee jerks were diminished and the Achilles' reflexes were lost. The Wassermann reactions of the blood and spinal fluid were negative. The psychotic features appeared at the age of 47, when he had two transitory attacks of excitement with much unclearness and a subsequent amnesia. It resembled the acute delirious states occurring in syphilis, that have been described by Plaut and others.

The author concludes that the tabes psychosis may occur in two types: one, an acute, transitory crisis-like disorder, and the other, as a more chronic, paranoid psychosis.

BARRETT, Ann Arbor, Mich.

LA NEVRITE HYPERTROPHIQUE DE L'ADULTE (HYPERTROPHIC NEURITIS IN THE ADULT). M. DIDE and R. COURJON, Rev. neurol. **26**:825 (Nov.) 1919.

On the basis of six personal cases and a careful study of the literature, the authors believe that the essential clinical picture, in this condition, may be described as follows:

The characteristic muscular atrophy seems to begin, consistently, in the upper extremities (very exceptionally the atrophy may begin in the lower extremities), and with very slow progression proceeds centrad, along the limb, from the periphery, until, finally, the entire segment may be involved. Signs of the tabetic series (ataxia, loss of sense of motion and position, hypotonus, etc.) are practically absent, and signs of the cerebellar series (dysmetria, intention tremor, asynergy, etc.) are but exceptionally observed and only in the very advanced stages. In connection with the latter, the authors question whether, in these cases, such reaction may not be wholly explainable on the basis of asynergy between unequally damaged muscular units rather than on actual cerebellar disturbance. The tendon reflexes remain intact for a long time, but, finally, may become diminished or even abolished. The idiomuscular reflexes follow the curve of the atrophy, appearing, at first, very active and later falling below the norm. No disturbance is noted in the cutaneous or ocular reflexes, nor is there evidence of nystagmus, strabismus, or exophthalmos. The electrical reaction depends entirely on the state of the muscles; but, in any case, it seems that reaction of degeneration appears only in the very late stages. Objective sensibility, as a rule, remains normal although a certain degree of hypesthesia and agnosia have been noted. The nerves involved are frequently painful to pressure, and dull pain and tingling are spontaneously noted along their course. Palpation reveals definite nerve hypertrophies which may be either diffuse or nodal in type. No disturbance is observed in genital function, sense organs or mentality.

In discussing the etiology of the condition, Dide and Courjon emphasize the fact that, from their study, there seems to be no hereditary or familial relationship nor any special predilection for age or sex. They believe that

neither syphilis nor leprosy are of any etiologic importance and suggest the possibility of some, as yet, undetermined infection with predilection for neuritic site.

Nosologically, the authors suggest the following classification:

1. Juvenile type, such as of Déjerine-Sottas and Marie-Boveri, which may be complicated by symptoms of the tabetic or cerebellar series.

2. Adult type, which may be of two kinds, namely, (*a*) simple type (as described above), and (*b*) complex type (rare)—simple type complicated by symptoms of the cerebellar series.

RAPHAEL, Kalamazoo, Mich.

INVESTIGATIONS ON THE CAPACITY FOR PSYCHIC FUNCTIONING OF THOSE HAVING HEAD AND BRAIN INJURIES, BY KRAEPELIN'S METHOD OF CONTINUOUS ADDITIONS. G. Vos, Allg. Ztschr. f. Psychiat. **75**:265 (April) 1919.

In this article is reported experimental work showing the capacity for psychic performance of a group of patients with head injuries, and following the progress of the disorders in their effects on psychic functioning. The author employed Kraepelin's method of continuous additions. This method offered possibilities of obtaining data for comparison of performance curves of those with brain injuries showing symptoms of more or less organic defects, with head injuries of the traumatic neurosis type.

The subject was required to add combinations of two numbers of one digit for a period of fifteen minutes. A rest period of fifteen minutes was followed by another fifteen-minute period of addition; a rest of ten minutes was then followed by a final addition period of fifteen minutes. The progress of the performance was divided among five minute phases, and the results were arranged in graphic curves. The author studied seventy-five cases of head injuries. In many of these the experiment was repeated at intervals of from four to six months. Of twenty-two subjects who had repeated the test, fourteen showed a greater or less improvement in their performance; in six it was unchanged, and in two the ability fell off.

These three groups of improved, unchanged and failing performance in repetition of the experiment all showed a striking agreement in their method of work, that is, the form of the performance curve. This seemed to eliminate any possibility of wilful alteration of the results in the anticipation of compensation for their injuries. The results of these experiments justified the following conclusions: 1. The greater number of those injured in the head or brain showed an improvement in their performance. A few showed a regression of performance capacity. 2. The results did not always agree with the progress of the physical symptoms of the subject. On the one hand, it was found that severe physical symptoms went hand in hand with good psychic performance, while on the other hand, with slight physical involvement psychic performance was poor. Improvement in the physical sphere did not always cause an accompanying betterment of performance ability. 3. The performance curve of patients with head and brain injuries showed marked individual differences. By repetitions of the experiment, the subjects held to a fixed type of performance, so that later curves were almost photographic reproductions of the earlier ones. 4. Most of the subjects showed at the beginning of the test an inhibition so that the performance of the first five minute phase was less than that of the second. 5. Most subjects had little ability to benefit by practice. Where this was marked it was usually associated with marked

fatigability. 6. The total performance of those with head or brain injuries was low, being about one third of that of normal subjects of the same intellectual level. 7. The larger number of cases of his series could be divided between two groups: (1) patients with marked organic disturbance and, (2) patients whose symptoms were largely functional (psychogenic). Between these two groups were many transitions so that a sharp separation was not possible. 8. The calculation ability of Group 1 was markedly better than that of Group 2. Poorest were the performances of a group of patients with traumatic neuroses of other origin, that had been studied for comparison. 9. Psychic effort in cases of head injuries may precipitate a convulsive attack. Therefore caution should be used in deciding as to the working capacity of those who have had head or brain injuries, especially in respect to those who have had epileptic attacks following the injury.

BARRETT, Ann Arbor, Mich.

LA LIPODYSTROPHIE PROGRESSIVE (PROGRESSIVE LIPODYSTROPHY). L. BOISSONNAS, Rev. neurol. **26**:721 (Oct.) 1919.

On the basis of twenty-two cases (two personal), the author concludes that the clinical picture in this disturbance may be thus standardized:

There is, fundamentally, a very gradual atrophy of the adipose tissue of the upper portion of the body, attended by fatty hypertrophy below the waist. The atrophy is first noted about the face, following which the neck, thorax, shoulders, arms and abdomen are successively involved. The mammary glands, however, are, as a rule, not involved. The skin itself shows no definite changes although there have been noted increased sudomotor activity and a peculiarly malodorous sebaceous secretion. In regard to the concomitant hypertrophy of the lower extremities, it has been found that the gluteal and gluteocrural areas are first involved, and in fact, may be the only areas affected, although in most cases this process later affects the thighs and legs, generally stopping at the ankles. The fat is deposited in a uniform manner, following limb contour, although occasionally, one limb may appear, as a whole, more hypertrophied than the other. The muscular and osseous systems show no disturbance. Sensation remains essentially unchanged, nor is there definite evidence of characteristic mental change. For the most part, cranial and peripheral nerve function remain undisturbed, although, in isolated cases, mention is made of exaggerated tendon reflexes, unilateral tremor, abolition of plantar reflexes, diminished pupillary reaction to light, nystagmus, facial tics and dermographia of the lower extremities. Boissonnas finds no evidence of relation to age or sex, but notes that this disease has been observed following infectious diseases and the establishment of menstrual flow. In discussing the etiology, the author feels that the essential cause is some, at present, more or less obscure lesion of the central nervous system and not, as some have suggested, primary endocrine dysfunction.

RAPHAEL, Kalamazoo, Mich.

NEOLOGISMS OF THE MENTALLY DISORDERED. S. GALANT, Arch. f. Psychiat. **61**:12, 1919.

This is a psychopathologic study of the peculiarities of speech so frequent among certain types of the mentally disordered. The few studies previously made on this subject have concerned the neologisms of dementia praecox. In the present contribution the author has studied not only those of that disease, but those of epilepsy, in much detail. His purpose was to find the ideas

that were back of the neologisms or neologism complexes and the origin of the peculiarly formed words. He believed that the neologisms did not appear suddenly, but had passed through phases of development, and that their content must represent ideas that had been much in the patient's mind in health or in the early years of his disease. His researches convinced him that most of the neologisms are not entirely senseless; that they are often ideas over which the patient has long brooded, and which are with a certain logical method expressed automatically in the form of neologisms. He attempts to demonstrate that there are types of neologisms characteristic of the different forms of dementia praecox and in this respect the form of neologism may be of diagnostic value. In the paranoid type the neologism occurs as a part of a system. It is intimately connected with the personality of the patient. His proposition is supported by a detailed analysis of the productions and life histories of two patients. The chief difference between the paranoid and catatonic type of neologism lies in the precipitating stimulus. In the paranoid type the stimulus that leads to the neologism is purely psychic and the neologisms are psychogenic. Those of the catatonic come from many stimuli; these are not psychogenic, but are from motor speech elements. In the paranoid type it is easy to follow the gradual development of the neologisms, but not so in the catatonic. The catatonic patient is not conscious that he uses new words; they come from the tongue and not the brain. Neologisms rarely occur in the hebephrenic type. These the author designates as symbolic. They are words of ordinary speech used symbolically and are not true neologisms. Although the foregoing three types of neologisms are well differentiated from one another, there are cases in which the types intermix or occur as transition forms.

Next to dementia praecox, neologisms are most frequently found in epilepsy. In outward form the neologisms of the two diseases are difficult to distinguish. The newly formed words of the epileptic patient are speech contaminations, abnormal combinations and peculiar usages of suffixes; while these occur in dementia praecox, there are differences in that the epileptic does not create new words except when they are in continuity with other words he is speaking. They cannot be understood except when interpreted in connection with the psychic peculiarities of the epileptic. They are determined in part by the impairment of memory, the circumstantiality, the tendency to form rhymes and to speak in symbols present in this disorder. The author presents a number of extensive thought productions of epileptic patients that are analyzed in much detail.

The neologisms occurring in other psychoses show nothing characteristic.

BARRETT, Ann Arbor, Mich.

THE POSITIVE WASSERMANN REACTION IN NONSYPHILITIC DISEASES OF THE BRAIN. FELIX STERN, Arch. f. Psychiat. **61**:725, 1920.

Plaut, Jahnel, Hauptmann, Mucha, Kramer and Zadek are quoted as having observed a positive Wassermann reaction in the cerebrospinal fluid in tuberculous and other meningitides not syphilitic. Plaut asserts that many of these cases occurred in syphilitic patients in whom the meningitis caused an undue permeability of the meninges and allowed the passage of the reagent from the blood into the spinal fluid, but Stern claims that too many cases have been reported in which the blood was negative and the spinal fluid positive

to permit much weight to be placed on Plaut's explanation of the phenomena and considers, therefore, that every unspecific reaction should be carefully noted. The cause of this unspecific reaction has been assumed by some observers to be the cell degeneration of the pleocytes and by others to be the alteration in lipoid substances, but no definite conclusion has yet been reached. The author urges a more critical study of all the reactions of both blood and fluid, and after noting that many observers have found a positive blood Wassermann reaction with negative fluid in nonsyphilitic tumors, he reports his own case of a combination of malaria with a small sarcoma of the cerebellopontile angle in which the Wassermann reaction of the blood was weakly positive and of the fluid + + + positive from 0.1 on.

An interesting point in the case was that treatment by quinin and such drugs always caused such great improvement of the ordinary tumor symptoms, such as vertigo, vomiting and headache, that it was only on the appearance of marked vestibular disturbance with deafness that tumor was suspected. Decompression was then practiced, and a radical operation was in preparation when the patient died. The tumor was the size of a small pigeon's egg. There was no leptomeningitis and no vessel alteration. The malaria was tertian in character, and the plasmodia were found constantly in the attacks which were repeated several times with a lapse of from three to nine months between the exacerbations. The diagnosis of a nonsyphilitic cause for the fluid Wassermann reaction was made first from the absolutely negative history of the patient, then from the failure of all syphilitic symptoms and finally from the peculiar reactions of the fluid itself, in which there was a marked increase in albumin with complete absence of pleocytosis, the paradoxical phenomenon found in different affections leading to increased pressure in the brain, but not found in syphilitic affections. A positive Wassermann reaction in the blood serum in malaria had been observed many times before the war, and the experiences of the war have shown that it is relatively frequent.

Stern considers the positive Wassermann reaction in the fluid in his case as "specific" for malaria, just as in syphilis it is "specific" for that disease, i. e., the reaction stood in direct causal relation to the malaria as it was not to be explained either by pleocytosis, which did not exist, or by reduction of lipoid substances as the tumor was very small and showed absolutely no degenerative changes. In all probability, the meninges were rendered more permeable by the action of the malaria permitting the passing of the complement reagent into the fluid from the blood. Whether the complement may be formed in the meninges is still unknown.

GURD, Ann Arbor, Mich.

THE CAUSES OF FEEBLEMINDEDNESS IN EARLY LIFE. SCHOTT, Arch. f. Psychiat. **61**:195, 1919.

This is a study of such causative factors of feeblemindedness as might be ascertained from the histories of 1,100 patients as given by their families. Appreciating the defects that are inevitable in such information, the author made a critical study on the occurrence of the following factors: (1) hereditary stigmatization including alcoholism of the parents and ancestors, (2) blood relationship and illegitimacy, (3) head and birth injuries, (4) brain disease and convulsions, (5) infectious disease, (6) rachitis and tuberculosis, (7) psychic traumas of the mother during pregnancy.

As regards the age from which the mental deficiency dates, he found that 60 per cent. of feeblemindedness may be considered congenital and 18 per

cent. as acquired during the first years of life. The remaining 22 per cent. resulted from factors active during the second to the fifth year, and a few developed in later years.

His conclusions are: As a general cause, diseases of the brain are of the highest importance in the production of feeblemindedness. These occur in at least one third of the series. Heredity and alcoholism of the ancestors is the sole cause in one third of the cases. In about one seventh of all cases no cause can be found. The remaining, about one third, are ascribable to a variety of causal relationships.

The most important medical problem in this field is to investigate and attack all pathologic processes that may act injuriously on the child's brain, either intra-uterine or after birth, particularly the infectious diseases. The campaign against feeblemindedness is intimately connected with the problems of hygienic and social betterment.

As special causes, hereditary stigmatization is of the greatest importance. In more than two thirds of these cases a moderate degree of feeblemindedness was present in the ancestors. Blood relationship and illegitimacy are not important causes. Head injury is of importance only when there are symptoms indicating severe injury, such as unconsciousness, vomiting and spasms. Birth injuries are in a similar class. Diseases of the brain are of much significance, and prevention of these will have an influence in reducing the number of feebleminded. Convulsions, which occur in about 8 per cent. as the sole cause, are to be regarded as due to brain disease. The conquest of the infectious diseases and the elimination of their causes will have a beneficial influence. Whatever can be done toward diminishing the amount of tuberculosis and rachitis will be of great value.

As there are in about one third of all cases several causal relationships, it becomes essential to direct the attack on feeblemindedness from various aspects.

BARRETT, Ann Arbor, Mich.

EIN WEG DAS WESEN DER "GENUINEN EPILEPSIE" ZU ERFORSCHEN (A METHOD OF STUDYING THE NATURE OF GENUINE EPILEPSY). PROFESSOR HAUPTMANN, Monatschr. f. Psychiat. u. Neurol. **48**:1 (July) 1920.

The author points out the serious limitations of our knowledge of genuine epilepsy. In searching for the cause we have to consider three factors: hereditary factors, metabolic disturbances and histopathologic changes.

The question of hereditary factors can be solved only by careful anatomic studies. These must include not only such gross changes as the heterotopias, but also the most minute developmental anomalies. If these studies are carried sufficiently far, he believes that no epileptic brain will be found entirely normal. Probably many nonepileptic brains will show similar pictures. Assuming such congenital anomalies as one essential, how can we explain the development of the disease with its convulsive seizures and psychic equivalents? The author believes that a second fundamental factor comes into play—a metabolic fault.

If we assume that faulty metabolism is essential to the development of epilepsy, we must determine whether this fault is primary, or, in its turn, secondary to degeneration of brain tissue; or whether epilepsy is the result of both factors, coexistent but independent. It is the possibility of a metabolic disurbance secondary to brain damage or brain anomaly that makes it difficult to determine the initial factor.

The author believes that we may reach a solution of the problem by a study of war material. Here we can rule out hereditary factors, at least in a large percentage of cases. We can study in such cases (1) what metabolic disturbances result from cranial lesions in general; (2) what metabolic disturbances result from convulsive seizures per se (to be worked out in jacksonian cases in which the seizures are of local origin); (3) what metabolic disturbances arise in the true post-traumatic epilepsies, that is, in those cases, not to be explained by local irritation, in which the attacks develop long after healing of the wound. We must determine what takes place during this latent period; and we must determine whether changes are occurring in the brain which render it more susceptible to irritation, or whether metabolic disturbances are developing which must reach a certain degree before they cause epileptic symptoms.

Hauptmann summarizes the possibilities with respect to the pathogenesis of genuine epilepsy as follows:

1. Congenitally abnormal brain—exogenic irritants.

2. Pathologic metabolic products—endogenic irritants. (*a*) Normal metabolic products. (*b*) Pathologic metabolic products. (1) Primary metabolic disturbance. (2) Secondary metabolic disturbance dependent on cerebral degeneration.

3. Normal brain—metabolic disturbance.

4. Primary, progressive cerebral disturbance.

As the author suggests, the problem is a large one. Just what phases of metabolism are to be studied he leaves to the discretion of those who undertake the work.

SELLING, Portland, Ore.

POST DIPHTHERITIC OCULAR PARALYSES WITH REPORT OF THREE CASES. M. LOMBARDO, Am. J. Ophth. **3**:747 (Oct.) 1920.

The author reviews the literature using an intensive bibliography. He reports three cases, the first of bilateral isolated paresis of accommodation and the second and third of isolated paresis of the right external rectus. In these cases the paresis manifested itself from four to seven weeks after the appearance of the primary disease. With the view of determining whether the usual treatment is of any avail, either to shorten the duration or in retarding the final result of the paralysis, no treatment was advised. The paralysis lasted from four to six weeks, and at the present time the condition of the muscles is entirely normal.

Remarks: These three cases and many others reported in the literature show that the paralysis of the eye muscles may be a sequel of diphtheria irrespective of the use of antitoxin and the age of the patient. Diphtheria may paralyze the eye muscles without producing paralyses of muscles of other organs. Postdiphtheritic paralyses of eye muscles pass away spontaneously.

Treatment has no influence on the course of these paralyses. In the writer's cases paralysis lasted from four to six weeks. This is the usual duration of post diphtheritic paralysis, as far as we know from the cases reported in the literature.

The eye muscles in postdiphtheritic paralysis usually regain their function entirely.

REESE, Philadelphia.

MICROGYRIA AND ITS EFFECTS ON OTHER PARTS OF THE CENTRAL NERVOUS SYSTEM. W. PAGE MAY, Brain 43:26 (May) 1920.

A brief case report is given with a rather detailed discussion of pathologic findings. The reader is referred to the original for the details.

The patient had a left hemiplegia in early childhood, which persisted to the time of his death at the age of 36. He showed mental deficiency which increased as he grew older, also epileptiform convulsions, which were likewise progressive.

The main pathology was arrest of development of certain parts of the entire central nervous system, which caused secondary and tertiary atrophy and resultant striking organic and functional changes. The right hemisphere was one third smaller than the left, the pyramidal tract was atrophied, the left cerebellar hemisphere and left half of the spinal cord were distinctly wasted. The cortex on the right showed shrunken convolutions and a decidedly lessened proportion between the gray and white matter. The basal nuclei were similarly affected, showing paucity of fibers and cells. The left hemisphere was normal in every way, and, contrary to expectation, showed no evidence of compensatory hypertrophy.

The author defends the name microgyria as truly representative of the condition, stating that the name does not involve the causal factor, yet indicates clearly the seat of the grossest change. The case differs from those reported by Mott and Tredgold in that their cases were either definitely cortical or basal, whereas this case falls into an intermediate grouping, being partly cortical and partly basal.

PATTEN, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY AND SECTION ON NEUROLOGY

Joint Meeting, Nov. 9, 1920

WALTER TIMME, M.D., *President*

POLIOMYELITIS. Presented by DR. ISADOR ABRAHAMSON.

Dr. Abrahamson called attention to the existence, almost in epidemic intensity, of a disease syndrome transitional in symptoms between lethargic encephalitis and poliomyelitis. He discussed the difficulties of diagnosis and the variability of the clinical manifestations of lethargic encephalitis. Lethargic encephalitis having died away until only chronic and remitting cases persisted, poliomyelitis had reappeared in epidemic form in Boston and subsequently in New York City. In the latter city, the board of health had already reported ninety-three cases, most of which occurred in October.

Undoubtedly, many of the cases in this epidemic of poliomyelitis showed a clear cut poliomyelitis syndrome. But besides such irrefutable cases, there were numerous others characterized by a common syndrome, which supplied, as it were, the missing link between the average syndrome of lethargic encephalitis and of poliomyelitis. These transitional or polio-encephalitic types were both mild and severe. Patients with mild cases showed nasal and pharyngeal catarrh with fever, malaise and implication of the facial muscles, sometimes with diplopia. The following are brief notes on cases of the severe type:

First Case.—A boy of 16 had pharyngitis, nausea and vomiting followed by diplopia. The same evening left facial palsy developed, followed by involvement of the left side of the tongue. The temperature rose rapidly. The next day, there was oculomotor involvement, paralysis of the left side of the face and tongue and of the motor fifth nerve; there were left palatal palsy and marked midbrain tremor of the upper extremities. The abdominal reflexes and the right knee jerk and both Achilles' reflexes were absent, the left knee jerk was diminished. There were no Babinski reflex or Kernig's sign, and no retraction of the head. The spinal fluid showed 66 lymphocytes. The diagnosis was bulbar and midbrain poliomyelitis. The patient died that night.

Second Case.—This patient was a boy of 9, with early diplopia and rigidity of the neck. There was right dropped foot; a left Babinski reflex and Kernig's sign were present. The patient was lethargic for forty-eight hours. The spinal fluid within forty-eight hours of the onset showed 149 cells, 90 lymphocytes and excessive albumin and globulin; Fehling's reduction. The patient recovered with the exception of dropped foot on the right.

Third Case.—A 2 year old child, whose mother had lethargic encephalitis with considerable residues, showed a poliomyelitis syndrome.

Fourth Case.—Four members of the same family had acute sore throat just before a fifth member, a boy of 15, showed diplopia, ptosis on the left, tremor of the extended right hand, paralysis, and later atrophy of the left upper extremity, weakness of the right side of the face and retraction of the head.

There were no evidences of Kernig's or Babinski's signs, no nerve tenderness or hyperesthesia. The patient's condition improved, with the exception of the paralyzed left upper extremity.

Fifth Case.—A woman of 39 was aphasic for four days. She lost power first in the right upper extremity and then in the right lower extremity. There was myoclonic twitching in the upper right side of the abdomen. The left pupil was larger than the right, rigid and ovoid. Seventh nerve weakness, left externus and internus, right externus and flaccid weakness of the left upper extremity, bilateral Babinski sign and marked midbrain tremor of the left upper extremity were present. The blood pressure, heart vessels, and urine were normal. The Wassermann test was negative. The spinal fluid showed old blood (from seven to ten days old) and 14 cells, all lymphocytes.

Dr. Abrahamson said that he had been in communication with Dr. Zingher of the health department, who had studied the spinal fluids in the reported cases of poliomyelitis of this epidemic, and he said that the vast majority showed a great excess of lymphocytes, excess of globulin and Fehling's reduction. Almost all had at the outset a catarrhal inflammation of the nose and throat and a few had gastro-intestinal signs. The same peculiarity was noted in lethargic and influenzal encephalitis.

DISCUSSION

Dr. M. Neustaedter said that the cell count, globulin and albumin content of the spinal fluid alone without the clinical findings were no index of any particular disease. In any inflammatory condition of the meninges, irrespective of the etiology, there are alterations in the spinal fluid, in its cytology as well as its chemistry. Dr. Neustaedter said that his experience and the literature showed that early in poliomyelitis there was marked lymphocytosis and that later polymorphonuclears predominated. Reports of other investigators showed that with the Lange test about 45 per cent. of the cases of poliomyelitis gave the syphilitic curve. In his work on complement fixation of spinal fluids with poliomyelitis antigen he had obtained some gratifying results in poliomyelitis and recently also in lethargic encephalitis. Dr. Neustaedter thought that this specific complement fixation test should be tried more extensively and that there might be a specific reaction pointing more definitely to the etiology of the two diseases. He stated that he had conducted neutralization tests with poliomyelitis virus and the serum of recovered lethargic encephalitis patients since last March. This work had not yet been reported. The results showed that there was a close point of contact between poliomyelitis and lethargic encephalitis.

Dr. David J. Kaliski (by invitation) said that he was not convinced of the value of the complement fixation test for poliomyelitis, using the antigen perfected by Dr. Neustaedter. Most of the serums sent by Dr. Neustaedter were negative, while a few of the controls (not cases of poliomyelitis; most of them old specific cases or other neurologic conditions) gave fixation with the antigen. Therefore the question of the specificity of the antigen was to be considered. Using the organisms of lethargic encephalitis isolated by Loewe and Strauss as antigen in a complement-fixation test for this disease, he had not been able to decide definitely as to the value of the reaction, principally because of a falling off in the cases of the disease when he was prepared to study the problem. The work was proceeding and would be reported later.

Dr. Kaliski stated that there were no definite diagnostic criteria in the cerebrospinal fluid which served to distinguish poliomyelitis, lethargic encephalitis and the polio-encephalitis types. While there was likely to be a larger number of cells in the fluid in the former condition, with increased globulin reaction, and even faint opalescence of the fluid due to a large number of cells, this was not diagnostic, but merely suggestive. Hemorrhage and even xanthochromia had been seen in a few cases of lethargic encephalitis. The curves obtained with the colloidal gold test were not characteristic of poliomyelitis. A rare case had been reported showing a curve similar to that of paralytic dementia or syphilis, but he had not been able to confirm this in any of his cases. In encephalitis likewise no characteristic or constant deviations from the normal were found. In a few cases of superior poliomyelo-encephalitis, no changes with the colloidal gold test were found, while the lymphocytes were usually only moderately increased, rarely above 50 to 100 cells to the millimeter.

Poliomyelitis, encephalitis, poliomyelo-encephalitis and tuberculous meningitis frequently gave similar findings in the fluid as regards the cells, the globulin and the presence of sugar reducing substances, and the deviations were usually in degree only. Of course, in the last disease (tuberculous meningitis) the finding of tubercle bacilli cleared up the diagnosis. From the standpoint of the pathologist, the diagnosis of any of these conditions with certainty was impossible, with the exception of tuberculous meningitis.

Dr. J. H. Leiner spoke of four patients at the Lebanon Hospital on the service of Dr. William Leszynsky, in three of whom there were facial palsy and meningeal irritation that later proved to be of poliomyelitic origin. One child was somnolent and had no Babinski or Kernig's sign; knee jerks were absent. The patient died within forty-eight hours. One child, with facial involvement, gave a history of vomiting and gastro-intestinal disturbance three or four days before coming to the clinic. The illness then assumed a poliomyelitic character.

Dr. Abrahamson expressed the hope that by calling attention to the current epidemic, the symptoms and signs of which seemed to fall between the known varieties of poliomyelitis and encephalitis, new data might be collected and the relationship between influenza, lethargic encephalitis and poliomyelitis might be established.

PUPIL DILATATION AND THE SENSORY PATHWAYS. Presented by Dr. Joseph Byrne.

Dr. Byrne considered the mechanism of pupil dilatation as set down in the textbooks unsatisfactory. Experimenting with cats, Dr. Byrne found that after section of any considerable portion of the afferent pathways, pupil inequality ensued. Thus after section of one sciatic nerve or of the lumbosacral nerve roots, the contralateral was smaller than the homolateral pupil, whereas after section of the brachial plexus, or of any of the cervical nerve roots, as well as after hemitransection of the spinal cord in the upper or lower cervical region, or in the lower thoracic or upper lumbar region, the homolateral pupil was smaller. From eight to twelve days after section of one sciatic nerve (or other of the enumerated operations), intravenous injection of epinephrin caused the relatively constricted pupil to become, for a

time, larger than its fellow. This phenomenon is known as paradoxic pupil dilatation and Dr. Byrne's experiments were the first in which paradoxic effects have been observed after lesions of the afferent pathways. All previous observations had been made after lesions of the efferent pathways, that is, of the cervical sympathetic nerve. Besides anatomic section, Dr. Byrne had injected alcoholic solutions into the sciatic nerve and into the nerve trunks of the brachial plexus. The results corresponded closely with those obtained after anatomic section.

From the effects of complete cord transection at various levels above and below the upper thoracic region (inferior ciliospinal center of Budge) on the pupillary phenomena noted after section of the afferent paths, it was concluded that in the normal waking state afferent impulses from all parts of the periphery are constantly impinging on the cells of the lateral gray column in the upper thoracic region on both sides. These impulses determine the efferent flow along the cervical sympathetic nerve which keeps the pupil dilated. There is, however, another factor in pupil dilatation that depends on inhibition of the sphincter muscle. But this forms the subject of a separate study. Dr. Byrne's experiments give no support to the assumption of a hypothetic cerebrospinal dilator pathway running from the region of the pons to the lower ciliospinal center. In no instance had Dr. Byrne been able to obtain effects on the pupil after hemitransection of the cord in the cervical region which could not as readily be obtained after posterior nerve root section. However, hemitransection of the cord at about the level of the second cervical root caused marked homolateral pupil constriction. This, in Dr. Byrne's opinion, was in part the result of injury of the root pathways of the fifth cranial nerve and not the effect of lesion of any special cerebrospinal dilator pathway. Dr. Byrne thought that his experiments opened the way for a more exact interpretation of pupil inequality as encountered clinically, for example, in tabes, etc., whereas they seriously brought into question the value of the so-called Klumpke-Déjerine sign, which was usually interpreted to mean injury of the efferent cervical sympathetic pathways.

DISCUSSION

Dr. Sherwin (by invitation) asked for information as to the structures on which epinephrin was supposed to act in eliciting paradoxic pupil dilatation.

Dr. K. Sellards Kennard (by invitation) called attention to the fact that pupil inequality was a somewhat common occurrence among cats and asked whether any steps had been taken by Dr. Byrne to eliminate possible error from this source.

Dr. Walter Max Kraus asked whether histologic studies had been made on the cells of the lateral gray column and on the dorsal nuclear column. It seemed as if the pathways involved in the pupillary phenomena were identical with the pain paths. The main afferent sympathetic paths were those of pain which probably crossed in the spinal cord after entering with the sympathetic nerve.

Dr. Thomas Hayes Curtin (by invitation) asked how the epinephrin was administered and what its effect was on the musculature. He spoke of various methods of producing dilatation. It might be produced by the effect of the administration rather than by the drug itself.

DR. BYRNE, in closing, said that the site of action of epinephrin in eliciting paradoxic phenomena was presumably the myoneural junctions in the iris. As in the case of the blood vessels, epinephrin had a two-fold action on the pupil, namely, a primary dilating action, followed by a secondary constricting action.

Replying to Dr. Kennard's question, Dr. Byrne said that in all cases careful preliminary tests had been made, and after such tests many animals had to be rejected. Replying to Dr. Kraus, Dr. Byrne said that the histologic studies had been made and marked changes had been observed, for example, after sciatic section, in the cells of the dorsal nuclear as well as in the lateral gray columns. The changes were evident on both sides but were more marked on the contralateral side in the case of the lateral gray columns (Budge's inferior ciliospinal center). Dr. Byrne agreed with Dr. Kraus that the paths involved in the pupillary phenomena were closely related to, if not identical with, the pain paths. The experimental studies were undertaken in the first instance with the hope of substantiating physiologically and anatomically Dr. Byrne's hypothesis as to the mechanism of pain.

A STUDY OF MOTOR APHASIA OF THE CORTICAL OR MIXED TYPE. Presented by DR. SAMUEL BROCK.

An American railroad mechanic, 40 years of age, had used alcohol to excess, formerly with frequent intoxication. Cigarets were also used to excess, but no drugs. The family history was unimportant; there was no history of head injury; venereal infection was denied.

While in France in army service about two and a half years ago, the patient suddenly fell forward unconscious. On recovering, speech was affected and the right arm and the right side of the face were paralyzed. The face and arm had practically recovered, but the speech defect had improved little. Since the onset he had had seizures of a jacksonian type, beginning in the right side of the face and involving the right arm and leg. Examination revealed motor aphasia. The gesticulations, mimicry and slight irascibility seen in this type of speech difficulty were manifest. Stereotyped phrases were used, such as "just the same," "sometimes pretty good, sometimes I could do nothing," "If I come here, I'll have to give it up," "if you can," "Boston," "I cannot move." He was able to count up to seven and to speak the first two letters of the alphabet.

When asked to write his age he wrote 1881, the year of his birth. His replies were not the exact answer to the question asked, but closely related to it; for example, "when" seemed to be mistaken for "where." He was able to write few words from dictation, and could not write simple sentences or numbers of more than four figures. He appreciated his inability and noticed his mistakes. He was able to write his name. Copying printed or written words was done quite well. Reading printed or written words was accomplished slowly but correctly. He recognized all objects, and gave no evidence of apraxia.

There was no gross impairment of motor power. No Babinski or allied reflexes could be elicited. They were all exaggerated and equal on both sides, except that the right Achilles' reflex was slightly more active than the left, and at times the right knee jerk predominated. In the sensory sphere, an area of hypesthesia was noted, together with analgesia on the anterior and posterior portion of the right side of the chest and adjacent neck, down to the

level of the abdomen. There were no ataxia or adiadokokinesis and no Gordon-Holmes phenomenon. Tremor of both hands was present.

He had suffered from dyspnea on exertion, cardiac palpitation and precordial pain. The evidence of cardiovascular disturbance and the sudden onset postulated a vascular accident in a branch of the left middle cerebral artery supplying the posterior part of the left inferior frontal convolution and adjacent motor area. The residuum was considered to be either a cyst or an old area of softening. An operation was proposed with the understanding that although there was only a slight possibility of improving his condition, there was a possibility.

OPERATIONS AND FINDINGS OF ABOVE DESCRIBED CASE. PRESENTED BY DR. K. WINFIELD NEY

Under local anesthesia, a large flap was turned down in the left parietal region. About 7 cm. above the external auditory meatus a large cyst was found. Vessels were ligated around the cyst. During the ligation a large amount of fluid escaped from the cyst, after which it collapsed, leaving a cavity about 2 cm. wide and 1 cm. deep. The thin upper wall of the cyst was resected leaving a cavity that had the characteristic yellow appearance of degenerated brain tissue. The lesion had been located accurately.

About a year ago, seventy or more craniotomies performed under local anesthesia were reported before the Society by Dr. Ney, and since that time he had added about twenty-five additional cases. He stated that he was becoming more confident of the value of this procedure. The majority of neurologists were somewhat skeptical in regard to intracranial operations because of the high mortality. In this series of operations under local anesthesia there have been no operative deaths. The local anesthesia never had to be supplemented by a general anesthetic, nor had it been necessary to discontinue any operation because of pain, shock or any of the disturbing factors of general anesthesia. Blood pressure seldom changed during the operation. There was no pain unless the dura was pulled or too greatly traumatized. The vascular instability associated with many brain operations, such as edema, etc., had not been a complication following invasions under local anesthesia. The infiltration of the tissues gave an almost bloodless scalp incision which was also helpful in the elimination of schock. The prospects of intracranial surgery under this form of anesthesia, Dr. Ney felt, were most encouraging.

DISCUSSION

Dr. Neustaedter asked concerning the results of the operation. Had the jacksonian attacks been affected?

Dr. Timme asked whether any nystagmus had been present.

Dr. Byrne said that Dr. Taylor had been using local anesthesia at Fordham, and that he personally could not speak too highly of its advantages. The neurologist, incidentally, was given an opportunity to test cortex sensibility.

Dr. Brock said that the patient was no better. The jacksonian seizures were just as frequent as before, and the aphasia was the same. There was no nystagmus before or after operation. The findings of this case bore out earlier studies on aphasia. A cortical lesion at the entering and leaving pathways, caused a mixed aphasia, which was of distinct value from a localizing standpoint.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Nov. 18, 1920

EVERETT FLOOD, *President, in the Chair*

ORGANIZATION AND FUNCTIONS OF THE NEUROLOGIC DEPARTMENT OF THE MASSACHUSETTS GENERAL HOSPITAL. DR. E. W. TAYLOR.

Dr. Taylor spoke of their successful organization based on an "integrating policy." The principal factors were a larger personnel, special assignments for individual research and for intensive clinical work in a limited field, intimate association with other departments of the hospital and close alliance with the medical school of Harvard University. Mention was made of the importance of the social service department.

A final necessity of a well-organized clinic is a clinic secretary whose duty it is to see that the clinic runs smoothly and effectively, that the patients are attended to and that the needs of the physicians are met.

RESIDUAL SPINAL CORD SYMPTOMS FOLLOWING INTRASPINAL ANESTHESIA. DR. W. E. PAUL.

Dr. Paul reported two cases of loss of sphincteric control immediately following intraspinal anesthesia. In both at least partial anesthesia of the saddle back area existed.

The first patient was a single man, 50 years of age, operated on for fistula in ano in September, 1913. He passed his urine the first day after operation, but the second day and for fifteen months thereafter a catheter was required. When the dressings were removed he was found to have fecal incontinence. There was a typical saddle back area of anesthesia except that the skin of the penis was not involved and the scrotum was less anesthetic than the peri-anal and posterior thigh regions. The blood Wassermann reaction was negative. Knee jerks were present but the ankle jerks were not obtained. The pupils were equal and reacted normally. In August, 1915, the bowels were constipated and a laxative was required, but care was needed to prevent accidents. The patient felt his sphincter was improving a good deal. After fifteen months a catheter was used only once a day and after the twentieth month only three times. He noted, however, some escape of urine at times and on passing urine the amount was small and the urine would start and stop. The anesthesia persisted over the saddle back area twenty-three months after it first developed. For a year he had little, if any, sexual feeling, but after a year there was some return with good erections.

The second patient was a married man, 45 years old, operated on in the middle of June, 1920, for double inguinal hernia. He had retention after the operation and required a catheter. Constant drainage was resorted to for a time, and he left the hospital at the end of three weeks. Possibly he had some trouble in controlling gas immediately after the operation, but there was no incontinence of the bowels. About two months after the operation he had involuntary urination, especially at night. Four months after the operation he was using a catheter twice daily but passed considerable urine voluntarily with effort and straining. He had urgent desire to urinate when urine accumulated but after the catheterization was comfortable for about three hours. He

had involuntary passage of urine every night. The blood Wassermann reaction was reported as negative. The knee and ankle jerks were normally present. The abdominal reflexes were lively, but the cremasteric reflex was absent. The skin around the anus was anesthetic in moderate degree, but the scrotal and penile skin was not. The sexual function was preserved in good measure.

Dr. Paul refrained from attempting to state what the lesion might be. It did not seem likely that the needle or trocar caused the lesion as the symmetrical distribution of the anesthesia would not be likely to be due to direct trauma from the needle.

DISCUSSION

Dr. George G. Smith stated that he considered spinal anesthesia very valuable. It can be used when ether will almost certainly be fatal. The occasional bad results should be balanced against the probably fatal results from ether. In urologic work, spinal anesthesia is particularly useful; it is practical in cases of retention from stricture, especially when there has been an overdistended bladder for a long time and the kidney function is below normal. In such cases ether would almost certainly be fatal, yet with spinal anesthesia the stricture can be cut and the patient can begin immediately to drink water. It is difficult to explain such results as Dr. Paul has reported. The needle entering a vein and causing hemorrhage might have something to do with it. Such instances as the use of too hot a solution of an anesthetic may be passed over. It is possible for those things to happen but not probable. As to the toxic effect of the drug used, one might like to know what drug was used. Procain is preferable, and in a 2.5 solution, that is, a 5 per cent. solution diluted with an equal amount of spinal fluid, would undoubtedly have no toxic effect on the nerve substance itself. It is possible that tropacocain, which was formerly used, would be a little more toxic. It seems unlikely that there could have been complete sensory paralysis since, if there had been, the patient would have had no sensation from his bladder as it became filled.

A NOTE ON CERTAIN ELEMENTARY VISUAL HALLUCINATIONS IN THE PRESLEEP STATE. Dr. Sidney Lord.

The word elementary is perhaps not happily chosen in designating the varieties of sense experiences in question, for it seems to be used in different ways; for instance, in specifying the origin of the experience in the sense organ itself in conditions of clouded consciousness, or as denoting the fact that such experience is elementary in duration and constancy, insignificant in influencing behavior or in causing any falsity of belief; or elementary in elaboration or completeness, that is, elementary in the sense of being unorganized rather than organized.

The phenomena in mind are elementary as concerns resultant behavior or deception of the intelligence, but are not truly elementary in the physiologic sense; that is, they are elaborated sensory images and are called hypnogogic hallucinations, hypnogogic illusions, perception phantasms or visions. Though they are sufficiently well known to receive the colloquial name of "faces in the dark," they are perhaps never entirely normal and, if almost invariably benign in prognosis, have not always been regarded as free from possible sinister import. There is not a large recent literature. These phantasms are not therefore frequent problems for the clinician. Occasionally, however, they are so often repeated or accompanied by such perturbation of mind as to

significance and course, occur with such vivid objectivity, and, at the moment, a fear so harassing that they compel more than the usual scant attention accorded them by the consultant. Moreover, when these phenomena are observed critically, the conviction is forced on one that they differ in various ways. There seems to be no doubt that some are pathologic and that some are not.

CASE 1.—A woman of about 30 was in a state of profound nervous exhaustion. Her illusions at bedtime were elaborate landscapes. There was no doubt in her mind as to their unreality, but there was a general anxiety about the presence of these illusions.

CASE 2.—A woman of 80, of alert and remarkable mind, but highly neuropathic, had from time to time been seeing ugly faces when the eyes were closed at night. No especial emotion accompanied these, or at least none was mentioned.

CASE 3.—An Armenian, about 50, presented himself complaining of insomnia and haunting visions at bedtime. He was intelligent, quite sane, sad, and much disturbed by the presence of these phantasms, profoundly so at the moment of their appearance, asking himself, "My God, why has this evil thing come to me? I am not crazy. What is it?" He described what he called "bad heads," "never a good head," and said, "The mouth keeps opening." He said the picture was always of a man or an animal picture. Asked what kind of an animal, he answered, "Never any kind that I saw before," except that the vision was, rarely, that of a serpent. This production of an image of a partly imaginary type is rather unusual. At least some of the images were gross exaggerations of anything in experience. He usually saw only the heads and not the other parts of the animals and men. They were not stationary; they always moved in one direction. The vision disappeared when he opened his eyes, to reappear on again closing the lids, but in "always different" guise. Sometimes it came when his eyes were open and it vanished when he shut them. He saw it moving away, and though he knew it was unreal, he was afraid; he pulled up the coverlet to try to hide from the horrid thing, closing his eyes to blot it out. It was grotesque, mysterious, unnatural; he was "awful afraid" but never deceived in his estimate of it. He tortured himself with the query, "Why does nothing beautiful ever come to me like this?" He never saw these things in the day, and never in the middle of the night; always at the first coming on of slumber. Graphically he stated that they were like moving pictures—never any noise. Asked why they come, he said, "I don't know why they come; they come themselves." Again, "Sometimes I watch it and then anything I remember comes." This last was a slightly different state suggesting the childish power of voluntarily evoking phantasms.

CASE 4.—A man of 50, intelligent, sane, neuropathic, had been subject for years to a variety of sensory shocks in the predormitium, to use Weir Mitchell's phrase. The visual images were usually seen at times of overwork and exhaustion. One type was the grotesque face which appeared with opening and shutting mouth. It was described as an unpleasant experience, accompanied by a general feeling of mental distress. Opening of the eyes dispelled the vision, but it tended to recur once or twice before he went to sleep. This was the common type. A much less frequent occurrence in this person was that of an extraordinarily vivid visual hallucination of scenery, of landscape. This occurred just before going to sleep when the senses were off their guard, but with the fully retained power of opening the eyes instantly to dispel the vision, which began with rudimentary, indistinct outlines of an object. Strikingly

like a developing photographic plate this vision expanded rapidly in detail and assumed the aspect of a landscape, surpassingly vivid and complete, indescribably clear and actual in appearance. The perception seemed to be one of transcendent brilliancy, vividness and beauty, quite beyond the realm of experience. It therefore seemed unnatural and awful. Fear arose and seemed to become overwhelming as the picture acquired a quality of being too real to be bearable. The patient felt as though he could put out his hands and pull out of the picture the objects which he saw. This was all so manifestly incongruous and false as to arouse the fear of something wrong and sinister—and the vision was then dispelled voluntarily by opening the eyes.

Such sensory impressions are different from dreamstuff. More nearly allied to dreams seems to be the vaguer imagery arising under similar conditions without any accompanying feelings of inappropriateness or dread of the unknown. The typical dream image is accepted by the subject without definite protest, so to speak, whereas these vivid phantasms are recognized as such and not accepted. Freud affirms, in speaking of hypnogogic hallucinations in general, their continuity with the dreams of the true sleep following. Dr. Lord is inclined to doubt this in relation to the more vivid and fear-compelling illusions spoken of.

These illusions are reported in part to emphasize two things which are not urged in the descriptions so far noted, namely, the quality of ultrareal objectivity felt by the perceiver and the degree of alarm experienced at the moment of illusion.

DISCUSSION

Dr. A. W. Stearns spoke of the relation of the fears to the illusions. He believed that the emotion was primary, and advanced the argument that the person being afraid had the illusions rather than that the person having the illusions became afraid.

Dr. Lord stated that he had described the sensation of fear as it appeared to the person who had the emotions. To him there was a sequence in time. He saw the rudimentary vision begin without any emotional accompaniment. As it grew it was so unreal and extraordinary that it seemed to him to signify something sinister even though he recognized at the moment that it was a phantasm; hence, as he thought, the fear.

Dr. E. W. Taylor mentioned a case of hallucination in an hysterical young man. He had the following experience: He was aroused by hearing a sound as of knocking immediately followed by a violent explosion. This was so vivid that he arose, turned on the light, went to the hall door and looked out expecting to smell smoke. He found nothing and returned to bed. This experience was repeated three times, the explosion occurring in different parts of the room accompanied by vivid red flashes of light. The realism continued so that each time he felt confident of the actual explosion in spite of his previous experience. This same night, while the lights were out, he had the illusion that the furniture was being violently moved; this illusion was immediately allayed by turning on the light. He had not before, and has not since, had similar hallucinations.

SOME OBSERVATIONS ON THE EYE IN DISEASES OF THE NERVOUS SYSTEM. Dr. C. A. McDonald.

Eight cases were reported. Three patients had double choked disk. One of the cases was diagnosed as a cardiovascular renal condition. There was an unusual degree of choking for this condition, but a low functional kidney

test corroborated the diagnosis. Another patient with a milder degree of choked disk and cardiovascular renal condition had a trace of albumin; the blood pressure was systolic, 230, diastolic, 140, and the retinal vessels were tortuous. The blood and spinal fluid Wassermann reactions were positive and the spinal fluid pressure was 350. A third patient in this group had double choked disk with a spinal fluid pressure of 90 and central chorioretinitis and numerous vitreous opacities, but the Wassermann reaction of the blood and spinal fluid was negative under potassium iodid and mercury treatment. The patient has improved much, but still receives mercury treatment.

There were three cases of unilateral choking of the disk, two following injury to that side of the head. The other, in addition to the choking of the disk, showed chorioretinitis in the vascular region and spots on Descemet's membrane. The importance of an examination of the media was emphasized.

There was one patient with optic atrophy with negative blood and a positive fluid, who improved considerably after receiving intravenous arsphenamin treatments. Another patient had complete optic atrophy of the left eye, failing vision (20/40) and a pale nerve head of four weeks' duration on the right. The blood and spinal fluid were positive for syphilis. After five intravenous arsphenamin treatments given weekly, vision was 5/200.

EXPERIENCE WITH SPINAL CORD TUMORS DURING THE PAST TEN YEARS. Drs. George Clymer, W. J. Mixter and Hugo Mella.

Dr. George Clymer reported a group of cases occurring at the Massachusetts General Hospital since 1912, with nontraumatic lesions involving the spinal cord on which laminectomy had been performed or considered. In all of them the outstanding clinical picture was that of cord compression, with or without nerve root irritation.

The group consisted of fifty-two patients of whom thirty-seven were operated on; a lesion accounting for the symptoms was found in twenty-nine. One of the cases operated on with no findings at necropsy showed a tumor of the cord at a level slightly higher than that at which it had been localized. In four patients a lesion not found at first was found at a second operation. The final diagnoses in the remaining seven, in whom no lesion was found at operation, were: multiple sclerosis, myelitis, question of brain tumor, sciatica, cord tumor, question of syringomyelia and lateral sclerosis. Of the twenty-nine patients in whom a lesion was found, four were operated on twice, the lesion not being found until the second operation, or if demonstrated at the first operation, an attempt to remove it was not made until the second operation.

The symptomatology of the patients as they appeared in the hospital was: pain in thirty-two cases, numbness of legs in twenty-one, inability to walk or loss of strength or stiffness of legs in twenty-one, unsteady gait and ataxia in four, dragging of feet in three, limping in two, incontinence of urine in eight, incontinence of feces in eight (these two symptoms did not always occur in the same patient), urinary delay in two, constipation directly associated with the onset of other symptoms in two, constriction band occurring in three and spasmodic twitching of legs in one. These symptoms are not separated according to type of lesion because they seemed to occur rather indifferently in all types.

The age at the onset of the symptoms varied from 3 to 58, the average being 40. The duration of the symptoms prior to operation varied from three months to twelve years, the average being between two and three years.

The final results of operations are not at present available for the whole series. In eight cases, the lesion was removed at operation. Twelve patients had been relieved when discharged; fifteen were unrelieved and two died.

Diagnostically, pain is the most frequent symptom in lesions causing cord compression, but is not universal even in cord tumors. The next important symptom is a sensory level, but this also does not always occur. The greatest help in the diagnosis of these lesions has been given recently by Dr. J. B. Ayer in his use of combined cistern and lumbar puncture. (The foregoing is the outline of a preliminary report.)

Dr. W. J. Mixter spoke on surgical difficulties and described the technic of operation. The operative mortality should not be excessive but shock is to be expected. In a series of forty laminectomies he had had but one death, and that was due to operative shock. Another death, however, was that of a woman with malignant disease of the spine. A specimen was removed for diagnosis and a laminectomy was not performed. Death followed shortly.

The prognosis of spinal cord tumors depends on the type of tumor. Cases of intramedullary tumors have all resulted unfavorably. In only one case could it be removed, and then no improvement followed. Another type in which the prognosis is poor is metastatic malignant disease of the spine. Operation is justified for relief of pain, but the relief lasts only for a few months. Tumors arising from nerve roots and tumors of the dura respond to operative treatment. Another important factor is the position of the tumor in the canal. Those situated anterior to the cord are more difficult to remove and the danger to the cord is much greater. Tumors of the cauda equina can be treated more satisfactorily. Tumors of the dorsal region are the easiest to remove, those in the lumbar region come next and the cervical tumors are most difficult. The most recent advance in technic is the examination of the spinal fluid by combined cistern and lumbar puncture. This gives definite and positive information as to the presence or absence of block in the canal. It is of great importance in eliminating certain of the degenerative processes involving the spinal cord. Cistern puncture is a dangerous procedure in untrained hands. It should never be performed except by a person skilled in the procedure. This procedure is a definite advance, but should be used sparingly and with the greatest possible care and precaution.

Dr. Hugo Mella stated that in the cases reported by Drs. Mixter and Clymer a definite pathologic diagnosis was made in twenty-nine. The types varied greatly in their order of frequency. There were found recurrent metastatic carcinoma, fibrosarcoma, tuberculosis of cervical spine, endothelioma, neurofibroma, glioma, myeloma, dermoid cyst including cholesteatoma, cyst of cauda, enchondroma, adamantinoma, malignant leiomyoma, solitary tubercle and chondroma. Both intramedullary and extramedullary carcinoma were found. Sarcoma arose from either the vertebrae or meninges and usually invaded the cord substance. Kyphosis following tuberculosis of the cervical spine caused a "kink" in the cord which, if not corrected, may cause complete destruction of the cord. The solitary tubercle is difficult to locate in life and may break down causing transverse myelitis since they are usually intramedullary. Attention was called to the work of Mallory on the so-called endotheliomas. In his article in the *Journal of Medical Research*, March, 1920, he showed that the cells from which these tumors grow are a part of the arachnoid and should therefore be named arachnoid fibroblastoma as they "invade" the dura secondarily. Closely allied to this group are neurofibroma, fibroma molluscum or von Recklinghausen's disease, which may involve the nerve roots or the

cauda. The perineurium, according to Mallory, is probably not an extension of the arachnoid along the nerves, but an analogous differentiation of the mesenchyma around them and, if we accept these findings, should be named perineural fibroblastoma. Dermoid cyst, including cholesteatoma, does harm by causing cord compression. Adamantinoma was found in the region of the cauda. It is a rare tumor developing from remnants of the enamel organ and is usually found in the lower jaw. The cyst of the cauda was probably the result of an incomplete closure of the neural canal, the remaining sack being filled with fluid; these tumors are probably related to hydromyelia. These cases did not include those of trauma, gumma or abscess.

In general, the outstanding features of these cases from the pathologic standpoint is their remarkable diversity.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Nov. 18, 1920

DR. G. B. HASSIN, *President, in the Chair*

REPORT OF TWO CASES OF ENCEPHALOMYELITIS, WITH PREDOMINATING CORD SYMPTOMS. DR. PETER BASSOE. (This paper will appear in full in the ARCHIVES.)

CASE 1.—A Chinese laborer, 50 years old, had urinary retention and weakness of all four extremities. The paralysis was not complete and was combined with marked ataxia. Patellar and ankle clonus was present and there was considerable muscular rigidity, especially in the trunk. Sensation was normal; the spinal fluid gave a cell count of 15 and weakly positive Ross-Jones and Lange reactions. After an illness of a few months the patient made a rapid recovery. The case was tentatively considered one of epidemic encephalomyelitis, with marked but temporary cord changes.

CASE 2.—A woman, 45 years old, was taken ill in August, 1919, with sore throat and nose bleed, followed by stiffness and pain in the legs. Diplopia was present for a few days, speech was slightly bulbar, and mental dulness and confusion were pronounced throughout the course. After a few weeks a picture of transverse myelitis in the thoracic region was established. At first the leg reflexes were increased, but they soon disappeared entirely. The spinal fluid gave a cell count of 26, a weakly positive Ross-Jones reaction and a fairly strong Lange reaction. Urinary retention and bedsores dominated the picture toward the end. The patient died on January 25, 1920.

The brain showed no gross changes and no important microscopic ones. The spinal cord was so extremely softened in the upper thoracic region that smears were stained in order to demonstrate that no pus was present, but merely necrotic cord substance. Numerous Gitter cells and fat globules were seen, but no micro-organisms. Sections from the rest of the cord showed extensive destruction of the white fiber tract and abundant fat granule cells. The ganglion cells were remarkably well preserved. Degeneration of white matter to the degree of necrosis was the main feature, inflammatory changes being less prominent. The case resembled others reported as instances of post-influenzal myelitis by Harbitz, in Norway, and others.

DISCUSSION

PROF. ANTON J. CARLSON: What was the condition of the sphincters toward the end? A considerable part of the sacral cord was intact.

DR. BASSOE: Yes, and in spite of that there was complete retention of the urine all the time. I don't remember about the anal sphincter.

DR. GEORGE B. HASSIN: In reference to the second case designated by Dr. Bassoe as myelitis, it would be more proper to call the condition of the spinal cord myelomalacia, or softening of the spinal cord. Just as softening of the brain occurs from vascular disturbances, a similar condition may obtain in the spinal cord from thrombosis or embolism, the former being much more frequent. The question is whether we can call such a condition myelitis. The myelomalacia in Dr. Bassoe's case involved exclusively the dorsal region, while the cervical, lumbar and especially the sacral, regions merely showed signs of secondary degeneration—with the hematoxylin stain. Dr. Hassin was sure that more refined staining methods will show even in these regions some definite changes.

In reference to the first case of lethargic encephalitis, he would like to ask Dr. Bassoe whether he remembered the condition of the pupils, because there is no lethargic encephalitis without some changes in the cranial nerves, especially the third, sixth and sometimes the seventh. In all of his nine cases of epidemic encephalitis studied pathologically, there were found most typical changes in the midbrain. It has been pointed out also in the ophthalmologic literature that some ocular changes are always present in this disease.

The spinal cord may be affected in epidemic encephalitis, but it never shows foci of softening, or necrosis which, if present, denote myelomalacia, as shown in Dr. Bassoe's second case. In epidemic encephalitis the inflammatory phenomena are infiltrative, in myelomalacia the changes could be termed alterative. Dr. Bassoe's remark that his first case was a most peculiar type of myelitis is practically correct. It is a peculiar type in the sense that the main changes are confined, not to the vessels as in epidemic encephalitis, but to the nerve elements themselves.

DR. BASSOE: In regard to the pupils in the first case, the case of the Chinaman, they were unequal at first, and then became equal, and they reacted normally at all times when I examined him. I did not see much of him during the first four or five days, when he was in the surgical department, but when I first saw him the pupils were equal. There were no cranial nerve changes that I could make out.

REPORT OF AN EPIDEMIC OF MENINGOCOCCIC MENINGITIS.

DR. RALPH P. TRUITT.

THE PRECISE LIMITATIONS OF THE INFLUENCE OF THE MIND UPON THE BODY.

DR. MEYER SOLOMON.

The following schema gives a working classification of activities within the human organism:

Mind			*Body*	
1. Consciousness a. Active, critical. b. Passive, uncritical.	Ordinary Waking Activities	Efferent	1. Spontaneous motor activities, locomotor, proficient, so-called voluntary.	
2. Ideas, including desires, wishes, cravings, etc.			2. Visceral activities, vegetative, automatic, so-called involuntary.	
	Emotions		3. Chemical or metabolic activities.	
		Afferent	4. Sensory activities.	

Under the term "mind" I include the activities of consciousness, whether active or passive, and of ideas, including under the latter term wishes, cravings and desires as well as ideas.

Under the term "body" I include the activities of a spontaneous motor, visceral, chemical and sensory nature.

The problem is: the possible influence of consciousness and ideas on the spontaneous motor, visceral, chemical and sensory activities.

During the act of thinking bodily changes of all sorts occur, for while thinking we assume and maintain certain bodily attitudes with simultaneous changes in the spontaneous motor, sensory, vegetative and chemical activities.

The Possible Influence of Ideas.—Under ideas, besides including wishes, cravings, yearnings, etc., I include suggestion, simulation, hypnosis, will power or volition, imagination, and their ilk.

The influence of ideas may be: (*a*) immediate, occurring at the moment of ideational activity, or (*b*) remote, occurring later, circuitously, as a final result of ideation. Examples of remote effects of ideas are: insomnia and its dire results, all flowing out of worry; irregular habits of living and the sad effects dependent on personal hygiene and behavior, the result of training and education, which can be reduced to instincts, emotions and ideas. In this way the remote influence may involve all bodily levels—spontaneous motor, visceral, chemical and sensory.

The influence of bodily changes produced may be (*a*) permanent or (*b*) transient. Remote influence may sometimes, with other factors, produce permanent bodily changes (such as arthritis or tuberculosis from unhygienic living). But the immediate result is always transient, except in such rare cases as emotionalism leading to the onset of hemiplegia or angina pectoris, etc., in one susceptible to them.

The discussion, therefore, is confined to immediate, transient influence of ideas. This may be (*a*) direct—without any intermediate link (as when an idea or wish leads to movement of arm or leg); or (*b*) indirect, with an intermediate link, as when an idea or wish leads to sitting, standing, walking or running, and the latter activity is accompanied, simultaneously but secondarily and accommodatively by visceral and chemical activities.

The possible immediate influences of ideas, wishes, desires, cravings, etc., per se, which are always transient, but may be direct or indirect, are: (*a*) on the spontaneous motor, locomotor, voluntary or proficient system.

1. Ideas can immediately and directly lead to transient functioning of this system—as in ordinary so-called voluntary activity.

2. Ideas cannot lead immediately and directly to disorders of the locomotor system of a prolonged and continuous nature of the sort described under post-traumatic hysteria. Such functional conditions, not due to gross organic changes, are fatigue or exhaustion states due to microscopic changes, with a psychogenic (ideational and emotional) element superimposed in most cases; or they seem to be continuous but are really transient (coming and going), as the result of inaction with absence of real efforts to produce proper functioning in the involved parts due to blind continuation of an original fatigue state which has since cleared up but in spite of which the patient continues to act by misguided conviction as if the condition of fatigue or exhaustion were still present.

3. In the cases of organic paralysis, whatever power still exists in the partially paralyzed muscles may be developed to its greatest possibility by training, exercise and various physical measures.

(*b*) On the visceral system.

1. The visceral, vegetative, so-called involuntary or autonomic system can be affected immediately, transiently, but only indirectly as the result of ideas—by primary, initial, preceding, or at any rate simultaneous changes in the spontaneous motor system. Salivary and gastric secretion of so-called "psychic" origin seem to be exceptions to this rule.

2. There is no ideational control, immediately and directly, over the involuntary muscles of the internal organs (heart, etc.). Indirectly, however, through the intervention of ideas, the involuntary system, for example the heart's action, may be modified, immediately and transiently, by adopting, perhaps unawares, even for a momentary period, certain attitudes or postures, which involve the voluntary locomotor or proficient system first, as in the "emotions" (see below) only to be accompanied instanter by certain changes in the visceral vegetative system.

3. Permanent prolonged or continuous changes in the visceral system cannot be reduced by ideas, even indirectly, except in cases in which, as the result of ordinary activity or emotion, an organic condition already existing is lighted up (apoplexy, etc.). This would be a remote, circuitous result and initiated indirectly but not continued by the idea.

(*c*) On the chemical or metabolic system. Changes of this sort accompany the act of thinking and occur with changes in the spontaneous motor and visceral systems but are not produced, transiently or permanently, directly or indirectly, by ideas in the sense in which these words are used in this discussion. We have a certain control over them, in so far as we control the ideational, spontaneous motor and visceral activities.

(*d*) On sensory activities.

1. Ideas cannot evoke, in direct or indirect manner, transiently or permanently, sensory phenomena—feelings of warmth, cold, touch, pain, pressure, etc.

2. Only by bringing about peripheral changes as the result of ordinary activity or emotions can any such effects be produced, such as angina pectoris from fear or overwork, with mechanical irritation of the nerve endings and subjective appreciation (consciousness or awareness) of this.

3. By ideas leading to distraction of attention one may inhibit or repress the degree of awareness, consciousness or feeling of sensations of all sorts.

4. But as in the case of continuous or prolonged motor paralyses, so also continuous or prolonged sensory losses cannot be produced by ideas.

Power and Mechanism of the Emotions.—In what we call the emotions, we have reactions at various levels—ideational, locomotor, sensory, visceral and chemical with, at the same time, a varying degree of consciousness, awareness or feeling of certain of these changes taking place. In the emotions are combined both mental and bodily reactions. But hence, too, the views expressed in the foregoing on the limitations of the power of ideas hold true, for the difference between so-called emotions and ordinary voluntary activity, especially when the latter is pronounced and exaggerated, as in running, is in the motive or the mental state present at the moment. Otherwise the modus operandi is the same and the reaction in the visceral and chemical systems present in the emotions is produced not directly but indirectly by the mental state, and accompanies or is secondary to activity in the proficient systems which precede it, and which itself is directly produced by the first reaction.

In the emotions there occurs first a change in the mental state—consciousness and ideas, followed in order by spontaneous motor activity, visceral (including ductless glands) and chemical changes.

In the emotions there is no new type of human activity but merely a certain combination of the activities of consciousness, ideation, the spontaneous motor, visceral, sensory and chemical systems, with the special characteristic of a peculiar mental state or attitude which differentiates it from ordinary non-emotional behavior.

Aside from the attainment of a mood of well being and mental calm, obliterating unnecessary, destructive emotions, and permitting a joyful, forward-looking, constructive attitude toward life, the value, possibilities and therapeutic efficacy of all forms of psychotherapy, legitimate or illegitimate, are both physical and mental, for they (*a*) directly and immediately lessen the drain on consciousness, ideation and the spontaneous motor system; (*b*) indirectly but simultaneously and immediately, through control of the locomotor system, lessen the drain on the visceral and chemical system; and (*c*) indirectly and remotely but not immediately overcome the tendency to emotionalism, bad habits and evil results from prolonged continuation of such unhygienic habits of living.

DISCUSSION

PROF. ANTON J. CARLSON: Eight or nine years ago I was quite a regular visitor at the meetings of the Neurological Society and at that time I somehow began to feel that this group was more interested in diagnosis than in etiology. That may have been an entirely erroneous impression. I hope it was.

The subject of Dr. Solomon's paper is a comprehensive one. To do it justice one would have to hold several sessions. It includes, of course, many points that are entirely unsettled, many points which are controversial, on which one may not be dogmatic.

Dr. Solomon said he would discuss the subject primarily from the clinical point of view. This Society is a clinical group and having invited a physiologist to discuss the paper, you will have to take the consequences if he discusses the subject from a physiological point of view. Not that there is any essential difference in the final result. But suppose I draw the picture as I know the subject, physiologically, and you already have the clinical picture, so you can take your choice, or refuse both.

In the first place I think that the title of the paper is not a happy one. I do not accept the mechanistic dualism of the "mind" acting on the "body." And I have for years deprecated that not a few neurologists make a distinction between "nervous" diseases and "mental" diseases. If the "mind" or brain is not a part of the body, what is it? In the physiological vernacular the action of the mind on the body means the nervous coordination of body functions.

As we analyze these diagrams of voluntary movements, movements of the heart, movements of the visceral organs (so far as they are supplied with musculature) and secretions (so far as these are controlled by nerves), we are dealing with nervous processes, leaving out for the moment the factors of chemical control. We are dealing with nervous processes passing out from the brain, the cord and peripheral ganglions and so far as these are dependent on afferent impulses conscious or unconscious, we are also dealing with nervous processes. The interrelation, conscious and subconscious, in the brain of afferent and efferent impulses is likewise a matter of nervous processes; so that from the point of view of a physiologist "mind" or mental action is merely a question of the correlation between the various parts of the body as

mediated by the nervous system. "Mind" in this sense is merely the sum total of conscious nervous processes or ideas going on in the brain at any moment.

I do not like the term "mind" even as defined by Dr. Solomon, because there usually is about it an aura of something mysterious and intangible, something of the stuff that filtered into us from nursery tales and primitive religious instruction. I am afraid that most of us, when we speak of the mind as distinct from the body, picture not only abstract ideas, but add ethereal arms and legs and mouths and noses to that mind as well.

But it seems to me we are on solid ground when we focus on the nervous processes. I do not offer this as the final analysis, but certainly from the point of view of physiology, it is the only avenue of actual progress, as shown both by the past and the present.

Dr. Solomon brought out one question which is quite controversial; namely, the relation of the emotions to ideas—the etiology of the emotions. I am neither a phychologist nor a psychiatrist, and nine-tenths of you probably know more about the subject than I do, but this is the way it looks to me. Some of the emotions may be hereditary. Possibly the emotional phases of appetite, of hunger, of sex, may be essentially inherited. I do not admit that they are. They may be. The capacity to feel or experience emotions is, of course, inherited. But apart from these, the emotional part of life is simply a matter of memory of the kind of relations or experiences we have had in the past with the particular idea or nervous process that happens to be going on in our brain at any one moment.

Let me illustrate my meaning by an example. Assume that we see a snake before us. In people who have been bitten by snakes that may arouse anger or fear. People who have touched snakes and found them cold may shiver. People who have never come in contact with snakes, but who have heard tales of snakes from the nursery up, may have a feeling of nausea: it is an evil animal, associated with the fall of man, and what not. In a primitive child, the snake would draw curiosity or delight according to its color, pattern or mode of motion. To a person who knows snakes, I mean biologically, there is neither loathing, fear, anger nor shivering but possibly a little of the curiosity and delight of those who know nothing else. Hence, the kind or quality of emotions (what we ordinarily call the qualitative phase of ideas) depends on one's individual experience in the past; it is a question of memory, of linking up the past with the present nervous process. The controversial point in the matter is this: whether emotions can be looked on as a memory process alone, or whether it is necessary to have all the bodily components in the original experience present as causes or sequences of the current memory process. Now so far as I know, gentlemen, that question is not yet settled.

You are all familiar with Sherrington's dog. Sherrington selected a very emotional dog; emotional in pleasure and joy, as well as in anger, disgust, etc. This animal was, by transection of the spinal cord and section of as many afferent nerves in the head and neck region as was compatible with life, deprived of most of its proprioceptive and enteroceptive fields, and yet after all that depletion this fraction of a dog retained his original emotions in quantity and quality. That experiment is not conclusive, but it takes more than talk to nullify its weight. To say that it is necessary to have concominant with the memory processes of emotional tone the action of the cardiac, the secretory, the motor or inhibitory phenomena of the genito-urinary and the gastro-intestinal tracts, etc., that were associated with the cerebral processes initiating the memory complex, is merely labeling an interesting theory as the

proved fact. Do you not agree with me, that the question of the physical reality of so-called imaginary pains is, to say the least, an open one? This is the way the facts appear to me: The physical reality or intensity of any memory process, including so-called imaginary pain, depends on whether these are the sole brain processes for the moment or whether these processes are balanced by the brain processes of critical judgment. In the absence of critical judgment, as in the child, the insane, in hypnosis, or in dream states memory processes or "imaginary" situations pass as real experiences, probably with the body reactions of the actual experiences. Most of us can "concentrate" sufficiently so that "imagination" or the mere memory processes will evoke the thrills of pleasure or the nausea of disgust in experiences remote in years of time and miles of space.

Certain things are pretty well worked out in the subject matter of Dr. Solomon's paper; that is, the nervous relations and reactions that are concerned with our external environment. I mean the skeletal neuromuscular system of normal persons. I do not mean to say that we know as yet all the intricate complications that can come up in this system in functional disorders. The efferent side of the so-called involuntary system is also pretty well worked out, as you all know, thanks to Gaskell, Langley and numerous other workers in this field. We have taken one system after another and determined precisely the kind of motor nerves it has, and how they connect with the brain; the kind of secretory nerves and their paths, etc. Anatomically these things are pretty well worked out. Physiologically the story is less complete. It is a long step in advance to lay bare an efferent nerve and by stimulating it artificially find that it causes secretion or inhibition. But these efferent facts alone do not reveal the complex working conditions of these systems in the intact body either in health or in disease; but it is a great step to know the anatomic details of the efferent mechanism. That is what I mean by this field being fairly well worked out.

Regarding secretory nerves to the glands of the internal secretions there is much literature; but there are few facts. I do not want to be dogmatic, but in my judgment secretory nerves to any gland of internal secretion have not yet been demonstrated. Cushing's work on secretory nerves to the hypophysis fell down. The work of a number of investigators on the secretory nerves to the thyroid gland is in the balance. Ten years ago we thought that the presence of secretory nerves to the suprarenals was proved, but we are less certain of it today. It is an unknown field. The working of the afferent or sensory components of the visceral system is also largely a matter of conjecture. I have a feeling that this system plays important rôles in health and disease in man, and new facts established here may mean more to medical progress than the writing of many books.

I am grateful for this opportunity to listen to Dr. Solomon's paper. We do not agree on some points. That is neither here nor there, provided his theories lead to active investigation.

Now and then there comes a superman who can blaze trails of real progress in a confused field. But we have had too much systematization in this "mind and body" field and too little intelligent work. What I hope is that the author's interest and analytical skill will not end with charts and schemata but in constructive work on patients and animals. Maybe I am prompted a little by selfishness in this connection, as it is clear to me that the rate of completion of our knowledge of physiology of the nervous system depends largely on the scientific interest and industry of the psychiatrist and the neurologist.

Dr. Solomon: In regard to the use of the term "mind," I did not really wish to use that term. I tried to get away from it by saying that when I use that term, I refer to consciousness and ideas. The reason I presented this subject in a dogmatic fashion was, so to speak, to throw down the gauntlet on various disputed points.

With respect to what Professor Carlson has said about instinct and emotions not being inherited for the most part, I think that what he means was that although the fundamental emotions like fear are really inherited, the attachment of the emotion to any particular thing is acquired in the life of the person and is dependent on experiences.

Experiments by Pavlow and others, rather conclusively prove that secretory nerves to the salivary and the gastric glands exist.

I have endeavored to adopt the clinical point of view in discussing a complex and obscure subject. We need the laboratory worker, we need the physiological worker, and we also need the clinician, who will endeavor to apply in an interpretative and synthetic way, in so far as he is able, the knowledge we have already gained from the laboratory and the clinic. We need to take a stand on these questions.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 26, 1920

Samuel D. Ingham, M.D., *President*

A PATIENT WITH MYOCLONUS FOLLOWING EPIDEMIC ENCEPHALITIS. Presented by Dr. S. F. Gilpin.

C. H., aged 45 years, white, an American, with a negative family and past history, about the middle of March, 1920, was ill apparently with a light attack of influenza. He remained at work until March 28, when he had a severe pain across the chest, extending to the testicle and rectum. It continued for three or four days. He soon became delirious; he continued in this state for five weeks, during which time he had muscle spasms and for three weeks he had hiccough. The jerkings continued all over the body and were so severe in the chest and abdomen that he could not rest; this state continued until his admission to Jefferson Hospital, September 14. He had lost about 50 pounds in weight.

Physical Examination on Admission.—The tongue showed a fine tremor. The pupils were equal and reacted normally. The eyegrounds were normal. The deep reflexes were increased; the bilateral Babinski sign and ankle clonus were present. The patient's mind was clear. The Wassermann test of the blood and cerebrospinal fluid and the urine reports were negative.

At present he shows no increase of deep reflexes and the Babinski sign and ankle clonus have disappeared. His chief complaints are the muscle contractions and pain in the left leg and about the testicles and rectum. This pain is paroxysmal, sharp and stabbing and so severe that he cannot sleep. When free from pain he sleeps well, and he has gained considerably in weight. The contractions are general, including those of the muscles of the trunk as well as of the extremities. They occur about seventy-two times a minute; they are not synchronous with the pulse, and their severity has lessened greatly during the past month.

A patient with a similar case was brought into the men's psychopathic ward at the Philadelphia Hospital last winter with a diagnosis of psychosis because of his delirium. He presented moderate temperature and the same marked muscle contractions. The interesting problems in the patient are those of the myospasms, which are rather choreiform in type except in their regularity, and the sharp shooting pains which resemble those of tabes and are distributed like root pains.

A CASE OF BILATERAL EIGHTH-NERVE PALSY; SYPHILIS. Presented by DR. M. A. BURNS.

History.—W. J. H., aged 26 years, a fireman, had a negative history; he denied syphilis. The patient had been married five years and had no children; one induced abortion.

In January, 1920, the patient was exposed to venereal disease, but claimed that he was not infected. Late in February, 1920, he suffered from what his physician called influenza, from which he recovered. During this illness he had a rash which persisted for weeks. The blood Wassermann test was then strongly positive. On Sept. 22, 1920, the patient came to the neurologic clinic at Jefferson Hospital. His chief complaint was deafness of the right ear, of one week's duration, and deafness of the left ear of three weeks' duration. He was dizzy and wobbly when he walked and had occipital headache.

Examination.—There was deafness in both ears and a facial paresis on the right. Romberg's sign was present. The pupils were equal and regular but reacted sluggishly to light and in accommodation. The grip was good. There was no tremor nor atrophy in the upper extremities, and sensation and the reflexes were normal. He was ataxic in both legs. The patellar reflexes were increased; no ankle clonus or Babinski's sign was present.

Laboratory Findings.—On September 22, the blood and spinal fluid Wassermann were ++++. The globulin was markedly increased in the spinal cord, which had in addition 104 cells. Under treatment, which has been vigorous and associated with drainage of the spinal fluid on five occasions, the Wassermann test on November 10 was ++ and the cells in the spinal fluid were 24.

DISCUSSION

DR. W. B. CADWALADER said that Dr. Burns' case was particularly interesting because of the lymphocytosis in the spinal fluid following, or associated with, the attack of influenza. Dr. Cadwalader had seen cases in which there were present the signs of ordinary myelitis, but it was difficult to determine whether they were due to syphilis or influenza; lymphocytosis occurred in both diseases. Dr. Cadwalader thought Dr. Burns' case was syphilitic.

DR. GILPIN stated that the patient gave no history of epidemic encephalitis. In the majority of cases the spinal fluid in this disease did not show lymphocytosis. Considering the symptoms and result of treatment, there can be little doubt as to the diagnosis.

DR. T. H. WEISENBURG said he did not recall seeing bilateral eighth nerve palsies in patients as young as this one, but he had seen nervous involvement in many cases of syphilis within one year after the infection and in a number while the secondary eruption was still on the body. He had seen third and seventh nerve palsies and one bilateral facial palsy within a few months after infection.

Dr. Weisenburg said that he had at the present time a patient with well marked tabes, with the history of an infection at 24, who a year and a half later, while she still had an eruption on her hands, presented pains, disturbance in gait and a beginning optic atrophy. This type of case was unusual. Dr. Weisenburg thought in these patients there was present one of two conditions, either a special predilection on the part of the patient for the specific virus or, what is more probable, a spirochetal infection which had a predilection for the nervous system.

Dr. Charles S. Potts said that he had seen a rash in a case of epidemic encephalitis which covered the entire trunk and resembled very much that of measles. It lasted about a week and then disappeared. Dr. Mills had also seen the patient and had concurred in the diagnosis.

DEMONSTRATION OF A DEVICE FOR SUPPORT IN FACIAL NERVE PARALYSIS. Presented by N. S. Yawger.

A description with photographs of this device appeared in the Archives of Neurology and Psychiatry, December, 1920, p. 659.

A CASE OF ASTASIA ABASIA. Presented by S. F. Gilpin for Dr. F. X. Dercum.

L. M., a white man, aged 58 years, complained of vertigo when attempting to walk and of a dragging of the right leg. Seven years ago, while going to work he noticed a paralysis of the right side of the face; he was told that he had had a stroke. When presented he showed evidence of an old Bell's palsy on the right side of the face, with some contracture. Otherwise there was no evidence of organic disease. In attempting to walk he hesitated, shuffled his feet and acted as though glued to the floor. When he was able to start to walk, he walked well, except for some stiffness of the right leg. The paralysis of the right side of the face and a friend's diagnosis of a stroke had apparently been the suggestive factors in bringing about his present nervous condition which was considered functional.

A CASE OF MYOCLONIC LETHARGIC ENCEPHALITIS WITH UNUSUAL PATHOLOGIC MANIFESTATIONS. Presented by Drs. N. W. Winkelman and T. H. Weisenburg.

Clinically, the unusual feature consisted in the pains which were sharply limited to the head, upper limbs, chest and abdomen to the line of the umbilicus and never below. The pathologic findings were those which are usually present in this disease, but careful study of the spinal cord demonstrated that there was a lessening in the intensity of the pathologic process as the lower levels of the cord were reached, and that the spinal cord below the lower thoracic level was altogether normal.

The patient was a physician, aged 40, with a history of influenza in February, 1920, seen by Dr. Weisenburg, April 22, 1920. Shortly after having had the influenza the patient complained of dizziness, which was not very severe. About a month before he was seen, he had hiccough which lasted for three days. A week later he complained of headache, the pain being all over his head and in his ear. Two weeks before he was observed, he began to complain of twitching in various parts of the body, first in the arms, then in the legs and

finally all over the body. Soon after, pains appeared and were described as sharp and shooting, but were never present below the line of the umbilicus. Two days before death, he became delirious and had visual hallucinations. When examined by Dr. Weisenburg on the day of his death, with the exception of visual hallucinations, he was normal mentally and discussed his case with some knowledge of the disease, although he insisted that he would get well. The twitchings were present chiefly in the shoulders, chest and abdomen, but were not at all marked in the lower limbs. There was considerable diaphragmatic twitching. The twitchings were of the character seen in Huntington's chorea, but were much more rapid. The limbs were not weak but the deep reflexes were increased. There was no Babinski sign, and there were no ocular palsies. He was seen at 5:15 p. m.; the twitchings rapidly increased, and he died suddenly the next morning at 4.

DISCUSSION

Dr. Williams B. Cadwalader said he had recently had a case of influenzal myelitis. The patient was paralyzed both for sensation and motion below the level of the costal border, but after some weeks there was almost complete recovery. Hyperesthesia at first was intense throughout the trunk and lower limbs; it seemed to be analogous to the hyperesthesia and pain sometimes seen in poliomyelitis, for it was relieved by lumbar puncture. The exact cause of the pain was not clear, but it is reasonable to suppose that there had been much swelling and edema of the cord, like that found by Dr. Winkelman. On this account the posterior roots might have been pressed against the vertebral wall becoming irritated, and thus caused pain.

Gordon Holmes has reported cases of gunshot wound in which he found a great deal of pain or hyperesthesia that he attributed to intense swelling and edema of the cord caused by contusion from the impact of a bullet, although in some of his patients the roots also were injured. Dr. Cadwalader suggested that the same explanation for central pain, as described by Holmes, might be given for the pain and hyperesthesia seen in the early and acute stages of influenzal myelitis and poliomyelitis. After withdrawal of the spinal fluid, Dr. Cadwalader thought the pain subsides in a striking manner. The pain in the muscles associated with repeated involuntary contractions seen in the late stages of epidemic encephalitis was of a different character and separate origin.

Dr. S. P. Gilpin said that in his case the first symptom was pain of such intensity that it drove the patient to the hospital. The delirium developed within four days after onset of the pain, and the muscle spasms developed while the patient was delirious. He thought Dr. Cadwalader's explanation of the origin of the pain was good, but in his patient the pain was still very marked, although several months had passed, and the patient had almost recovered from the myospasm.

Dr. Weisenburg said that as to the assumption of a resemblance between the pains of poliomyelitis and encephalitis, he was of the opinion that there was not the slightest ground for this. He had made what is probably the only neurologic study of a poliomyelitis epidemic, in 1916, when he reported 717 cases. As a result he concluded that in every acute case of poliomyelitis there was present a limited amount of meningitis, and that the pains in the limbs were the result of this. In fact, one could distinguish the paralyzed limbs because they were painful on pressure. Such a condition does not exist in encephalitis. Besides, the pains described were of a different character. In

encephalitis they are sharp and shooting; in poliomyelitis, while there are occasional sharp pains, the patients chiefly complain of pain in the head and neck and on pressure over the limbs.

AN INTERESTING CASE OF MYELITIS. Presented by Dr. F. B. Marsh.

This particular case was one of the many conditions in which influenza evidently played an important part.

A white man, aged 55 years, an engineer, was admitted to the Pennsylvania Hospital, Sept. 27, 1920. He complained of wasting and loss of strength in the muscles of the arms and shoulders and jerking of the muscles of the same parts. He denied syphilis. About five years ago the patient had had a stroke of apoplexy. He fell over, not unconscious, but his right arm and leg were paralyzed and he was unable to speak. These symptoms lasted only twenty-four hours, but his arm and leg were weak for about two years. His condition was good until about two years ago when he developed influenza. On recovering from that disease he realized that his shoulder muscles and the muscles of his arms were greatly atrophied and were practically powerless. There were no other symptoms. His grip was good. About one year ago he noticed that various muscles of the shoulder and arm jerked at times, and that his gait was slightly unsteady, His general condition had been good.

Examination.—The face movements were normal. The pupils were equal and very irregular; the right reacted to light sluggishly, the left more promptly. The tongue protruded in the midline. There was no paralysis of the vocal cords and speech was normal. The neck showed muscular atrophy and loss of power in the posterior and lateral groups of muscles. The trapezius and sternomastoid muscles were fairly well preserved. The patient was not able to lift his head up slowly, but had to throw the head back and hold it there by shifting the center of gravity. There was complete atrophy of all of the muscles of the shoulder girdle including the biceps. The forearms and hands were fairly well developed with good power in them. Biceps reflexes were absent; the triceps reflexes were exaggerated, especially on the right. Twitching of the muscles of the shoulder girdle was noted at times. The lower extremities were well developed and slightly spastic. The knee jerks were much exaggerated, but equally so. An ankle clonus was present on the right. There was no constant Babinski sign. There was no disturbance to heat or cold, but pain sense was apparently diminished in the right axilla. Electrical reactions were absent in the atrophied muscles. The triceps reacted normally. The roentgen-ray report on the cervical spine was negative. The urine, blood and spinal fluid were normal except that the Wassermann test on the blood was weakly positive on two occasions.

Dr. Marsh thought the most probable cause was either influenza or influenza with syphilis. An influenzal myelitis could have developed during the influenzal attack which just antedated the onset of the symptoms. On the other hand, the influenzal attack might have lighted up a preexisting syphilis. The fact that the patient's blood on two occasions gave weakly positive reactions would favor the latter view, while the negative spinal fluid Wassermann reaction would be against it. The nerves supplying the paralyzed muscles arise from the second to the fifth cervical segments. The preservation of the sternomastoid and trapezius is explained by the double nerve supply—the cervical nerves and the spinal accessory. The spasticity of the lower extremities indicates that the myelitis was not complete but involved, either by pressure or infiltration, the lateral columns.

DISCUSSION

Dr. Milton K. Meyers thought that often a previous injury or disease might obscure the diagnosis in such a case. Among returned soldiers he had found four cases of amyotrophic lateral sclerosis or of progressive muscular atrophy, which in some of the cases were unsuspected by the claimants; in some the nervous disease was attributed to injury. In the present case there was a spastic condition of the lower extremities which might well be explained on the basis of the patient's previous hemiplegia. It seemed to Dr. Meyers that the condition of the upper extremity could be explained as a progressive muscular atrophy. Absence of sensory symptoms would indicate this.

Dr. N. W. Winkelman said this was a case in which localization was obtained not from sensory, but from motor symptoms. There was absence of biceps reflexes, with biceps atrophy, representing the fifth cervical segment, with preservation of the triceps reflexes, the sixth cervical segment, so that the lesion was above the sixth cervical segments. Since the muscles of the back of the neck were distinctly atrophied there must be involvement of the second, third and fourth cervical segments. The patient's answers as regards objective sensation were contradictory but there were sensory disturbances, though these were not as marked as the motor disturbances. The lesion extended from the second to the fifth cervical segments, even though the diaphragm was not involved, since the lesion was probably incomplete.

Book Reviews

THE PSYCHOLOGY OF FUNCTIONAL NEUROSES. By H. L. HOLLINGWORTH, Associate Professor of Psychology in Columbia University. Cloth. Price, $2 net. Pp. 259. New York: D. Appleton and Company, 1920.

This is an interesting and able work, based on the observations by the author of approximately 1,000 cases in U. S. Army General Hospital No. 30 at Plattsburg Barracks. After consideration of many theories, he concludes that the mechanism of redintegrative response affords an explanation for the reactions of the psychoneurotic patient. He describes redintegration as "that type of process in which a part of a complex stimulus provokes the complete reaction that was previously made to the complex stimulus as a whole." He then discusses normal and abnormal redintegration, and points out that there are three levels of response discriminated in the reaction from a given stimulus or situation: (1) the postural or cerebellospinal level, (2) the cortical level, and (3) the autonomic level. In the psychoneurotic patient, because of more or less mental inadequacy or stupidity and lack of sagacity, there is not a normal redintegration in the cortical, autonomic and postural levels, especially in the spinal and autonomic levels.

In the mental ratings of psychoneurotic patients, the author's data consist of intelligence tests of nearly 1,200 patients. The mental age ranged from 8.3 years, in cases of mental deficiency, to 13.2 years in psychasthenia. The epileptic and hysteric groups had a mental age of about 12 years. The mental age of the normal soldier is not over 14 years.

The author states that "it is clear that the difference in specific symptoms is correlated with a difference in mental level," and that the lower the intelligence level, the more liable the soldier is to display the so-called conversion type of hysteria, and the higher the scale of his intelligence, the more likely he is to become neurasthenic or psychasthenic. Officers, for example, rarely show hysterical symptoms, usually having the so-called anxiety neuroses.

The author concludes that it is this predisposition toward redintegrative conduct that constitutes or characterizes what may conveniently be called the psychoneurotic constitution.

Finally he summarizes his conception of the functions of psychologic service in a neuropsychiatric hospital: "Through the intelligence examination to throw light on the clinical condition, the complete diagnosis, the proper disciplinary measures, the military or civil serviceability, and the most effective and expeditious disposition of patients. Through the further analysis of such data to make at least a suggestive contribution to the study of the factors operative in the production of the psychoneuroses. Through the inventory of the patient's special aptitudes and educational equipment, to place him effectively for maximal therapeutic occupation. In a similar way to afford him aid in vocational adjustment and in the development of purposiveness and aim, in the effort to make of him a more balanced and a better adjusted personality. Through graphic records of the increments of functional capacity under physio- and mechano-therapy, to portray for his own encouragement and for the information of the physician the course of progress. Through inten-

sive individual reeducation to direct and stimulate the patient in his recovery from specific symptoms and disabilities, thereby improving his general morale and his attitude toward the hospital and the service. Finally, through special experimental technic, to demonstrate the degree of rehabilitation and the approach to complete recovery in particular cases, in exact and comparable terms."

When the war broke out psychologic examinations were instituted in various camps. Later the psychologic corps, which at first was part of the neuropsychiatric division, was made an independent unit, for the first time in the history of any army. In connection with this notice of Dr. Hollingworth's book, it may not be amiss to give the point of view of most of the neuropsychiatrists who had actual experience with the psychologic examinations, working more or less with the author and his associates.

In analyzing the data and conclusions of Dr. Hollingworth it is interesting to note that he had, in his 1,172 cases, 339 epileptic patients, about one third of the total number. He, of course, was not responsible for the diagnoses that were furnished him, but it developed that most of these so-called epileptic patients really had hysteria. In the author's studies the mental age of epileptic and hysteric patients was similar. This is pointed out only to show that some of Dr. Hollingworth's conclusions were based on incorrect data.

Medical officers in Plattsburg regarded the psychologic service as of considerable value in the aid that it gave them in the mental examination of patients, saving them a great amount of work. The chief value of the psychologic service, however, was that it had workshops and other methods of keeping patients busy. But it is worthy of note that comparatively few soldiers would go to the shops voluntarily; most of them had to be ordered there. After the armistice the psychologic service was practically of no value, for the soldier had only one object in view and that was to be discharged. All this is said not to disparage Dr. Hollingworth's work, but to give the reader the point of view of the soldier and medical officer.

The value of the psychologic service in the army varied in different camps. For example, in one camp, in which the psychologists recommended for discharge all those whose mental age was 9 or 10 years, careful inquiry by the neuropsychiatrists showed that a large percentage of these men, although they had low mental rating, were able to get along in their civilian occupations and to support themselves and their families. In other words, they were relatively adaptable and efficient. On the other hand, there were cases accepted by the psychologists with a mental rating of from 10 to 12 years, who were found to be definitely handicapped, so far as their ability to earn a livelihood was concerned; they gave a history of shifting from one occupation to another and being unable to support themselves; they manifested, to a large extent, many reactions common to constitutional psychopathic persons.

Psychologic work went to the discard, however, after the March, 1918, offensive of the Germans, for then it was a question of sending many men abroad as rapidly as possible. The further the war progressed, the less the psychologic corps gained in favor and by the army as a whole it was regarded as an encumbrance. It is questionable whether in a future war, a psychologic department would be found in the Surgeon-General's Office. Every soldier of any experience knows that either a corporal or a sergeant of a company knows more of the individual merit of a soldier and what particular thing he can do best, than would be indicated by the most expert psychologic examination.

No doubt a psychologic examination can give the mental level of a man, but it cannot indicate the fighting level, for mental age does not with any accuracy indicate the vital factors of temperament, moral attitude, industry, obedience and other qualities in the make-up of a good or poor soldier (or civilian).

FUNCTIONAL NERVE DISEASE. An Epitome of War Experience for the Practitioner. Edited by H. Crichton Miller, M.A., M.D., Formerly Medical Officer in charge of Functional Cases, No. 21, General Hospital, Alexandria; Late Consulting Neurologist, 4th London Hospital. London: Henry Frowde, Oxford University Press; Hodder & Stoughton, Ltd., Warwick Square, E. C. 1920. Price, $4.50. Pp. 208.

The chief value of this work is for the general practitioner for whom it was written. For the neurologist it is interesting and instructive as presenting today's British thought on the psychoneuroses. As the book consists of twelve chapters by eleven authors, each writing from his own point of view, there is a good deal of disharmony and the composite gives the impression of heterogeneity. Indeed, an oldish observer has a feeling of being present at the birth of English neurology—in so far as it pertains to the psychoneuroses. The various chapters (well written, clear and helpful) represent fairly well sundry stages and phases in the development of neurology heretofore passed through by other countries. The great war at once fairly forced functional nervous disorders on medical England, and with the bull dog's customary slowness, tenacity and ultimate effectiveness medical England tackled the job. Consequently, this little book contains a good presentation of the psychoneuroses of war in their various relations and as looked at from different sides. Every chapter is good, though here and there the work is unmistakably immature. The chapters on "Psychoanalysis," "Repression and Suppression," "Regression and the Mother Complex" will be interesting and illuminating for many who have not already delved into these things, and assuredly will give them new and useful ideas on functional nervous disorders. Chapter 12, by William McDougall, is masterly. His task was to summarize the preceding eleven chapter. This he does not do; nobody could. But in seventeen pages he gives a remarkably clear and reasonable exposition of the psychoneuroses, the principles which should govern their treatment and the mechanism of their recovery.

The assumption by the editor that what we have learned of war neuroses will be useful in the estimation and treatment of peace neuroses assuredly is well founded.

Fifty cents each will be paid for the April and May, 1919, issues of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.